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The Type a Behavior Pattern and Acth Response to the Stress of Cardiac Catheterization.

Marie Carmen Veitia

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Veltia, Marie Carmen

**THE TYPE A BEHAVIOR PATTERN AND ACTH RESPONSE TO THE STRESS
OF CARDIAC CATHETERIZATION**

The Louisiana State University and Agricultural and Mechanical Col.

PH.D. 1986

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**THE TYPE A BEHAVIOR PATTERN AND ACTH RESPONSE
TO THE STRESS OF CARDIAC CATHETERIZATION**

A Dissertation

**Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy**

in

The Department of Psychology

by

Marie C. Veitia

B.A., University of New Orleans, 1979

M.S., University of New Orleans, 1981

December, 1986

Dedication

For my father who inspired me

For my mother who guided me

and for the love of my husband that sustained me.

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Abstract

There is substantial evidence to indicate that the Type A Behavior Pattern is associated with an increased risk of coronary heart disease. Recent research has shown that Type As, as compared to Type Bs, demonstrate enhanced biochemical and cardiovascular responses to stressful situations. These findings have led researchers to postulate that physiological reactivity may be one of the mechanisms through which Type A behavior confers coronary risk. The present study was designed to investigate physiological and psychological reactivity in Type A and Type B cardiac patients exposed to a cardiac catheterization. The effects of drugs (i.e., none versus beta-blockers and calcium channel blockers) on the response to stress was systematically evaluated. Dependent measures, adrenocorticotrophic hormone (ACTH), systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), state anxiety (STAI-state), and Total Mood Disturbance (TMD), were obtained prior to cardiac catheterization (day 1) and 24 hours later (day 2). A 2 x 2 x 2 (Behavior Type x Drug Group x Day) analysis of variance with repeated measures on day revealed significant main effects of day for ACTH, SBP, and state anxiety. A significant main effect for drug group and a significant Behavior Type x Drug interaction was revealed for ACTH. A significant Behavior Type x Day interaction was found for SBP. An additional analysis investigating the responses of

extreme Type A and Type B subjects revealed significant main effects of day for HR and State Anxiety. The results of the present study failed to support previous research demonstrating that Type A subjects, as compared to Type B subjects, exhibit exaggerated responsivity to stress. Possible reasons for this failure to find significant A-B differences are explored. The major contribution of the present study was the finding, consistent with previous research, that the target medications were associated with substantially reduced responsivity to stress (as measured by ACTH) in Type A cardiac patients to levels consistent with the response of Type B cardiac patients. The clinical and research implications arising from this finding are discussed. Continued systematic evaluation of the effects of medications on reactivity is strongly recommended.

CHAPTER ONE

Cardiovascular disease is the leading cause of death in the United States today. More than 43 million Americans have some form of this disease. Almost five million Americans suffer from coronary heart disease (CHD), a form of cardiovascular disease (American Heart Association, 1985). Among the cardiovascular diseases, heart attacks, one of the manifestations of CHD, are the leading cause of death, accounting for 554,900 deaths in 1982 (American Heart Association, 1985). Cost to the national economy in 1985 has been estimated at 72.1 billion dollars, including costs for hospitalization, physician and nursing services, medications, and disability costs (American Heart Association, 1985). Cardiovascular disease ranks second to respiratory diseases with respect to days of bed disability and first among diseases that limit activity and cause the greatest number of hospital bed days (Levy, 1982). Recent trends in medicine and technology, and increasing public awareness, have resulted in some decline in cardiovascular mortality. Despite this, however, cardiovascular disease remains a problem of significant proportions (Levy, 1982).

Research examining the participation of behavioral risk factors involved in CHD has witnessed growing interest (Eliot, Buell, & Dembroski, 1982) especially because of the evidence from studies indicating that behavioral risk factors are significantly related to CHD (Rosenman, Friedman, Straus, Wurm, Kositchek, Hahn, & Werthessen, 1964; Haynes, Feinleib, & Kannel, 1980). There is

now a substantial body of literature demonstrating the Type A Behavior Pattern (Friedman & Roseman, 1959), characterized primarily by a sense of time urgency, easily aroused hostility, and competitive achievement striving, is strongly associated with incidence, and often severity of CHD.

Coronary Heart Disease

Coronary heart disease (CHD) is a clinical term used to describe that group of cardiovascular diseases in which the primary symptomatic manifestations are angina pectoris (i.e., chest pain secondary to an oxygen deficiency in a region or regions of the heart), myocardial infarction (i.e., a consequence of coronary artery disease in which irreversible cellular injury and necrosis occurs secondary to prolonged ischemia), and sudden cardiac death. Sudden cardiac death is defined as unexpected, witnessed death in a subject with or without preexisting heart disease, who dies in less than one hour following the terminal event (Kloster & Bristow, 1985). The major etiologic cause of CHD is atherosclerosis, an accumulation of fatty deposits in the coronary arteries that restricts blood flow (Willerson, 1982). Arteries are blood vessels that flow away from the heart and carry oxygenated blood to various parts of the body. The coronary arteries are those which directly provide oxygen and nourishment to the heart itself. The process of atherosclerosis will be considered in subsequent sections of this paper.

A long list of major and minor cardiovascular risk factors have been described from epidemiological studies. According to

Levy (1982), the major risk factors are age, sex, hypertension, cigarette smoking, diabetes, high levels of low density lipoproteins, and deficit levels of high density lipoproteins. Low density lipoproteins (LDL) are the major carriers of cholesterol. There is a positive association between high levels of LDL and CHD. High density lipoproteins (HDL) provide a protective effect that is as yet not clearly understood. The HDL level is inversely related to cardiovascular risk (i.e., the higher the HDL, the lower the cardiovascular risk). Hyperlipidemia, defined as serum cholesterol and/or triglycerides at levels higher than the 95th percentile for controls, is thought to be a major risk factor for atherosclerosis. A number of minor risk factors have also been reported. These are family history of heart disease, obesity, sedentary lifestyle (or lack of regular physical activity), and emotional stress (including personality type).

Aside from physical examination, several diagnostic procedures (e.g., chest x-ray, electrocardiography, echocardiography, cardiac catheterization) are available to the physician interested in evaluating cardiac function. The following discussion focuses on cardiac catheterization, because this is the procedure used in the present study. Cardiac catheterization (also known as coronary arteriography or coronary arteriogram) is a powerful diagnostic procedure that allows for comprehensive assessment of cardiac structure and function. It involves the detailed measurement of intracardiac pressures and

blood flow and the angiographic visualization of heart and coronary arteries (Harrison, 1982). A cardiac catheterization is employed when cardiac condition cannot be adequately established through history, physical exam, or the use of noninvasive techniques. It can serve primarily as a method for providing the anatomic and physiologic details necessary for planning surgery (e.g., coronary artery bypass surgery). It can be used to evaluate the severity of a known cardiovascular disorder in which there is a discrepancy between symptoms and physical findings. It can also be employed as a diagnostic procedure in the assessment of suspected cardiovascular disease or to clarify the presence of symptoms with unknown etiology. In the patient who complains of recurrent chest pain consistent with angina pectoris, the cardiac catheterization is a necessary and invaluable technique for establishing the presence of coronary artery disease (Wallace, 1982). It is currently the most reliable diagnostic test to detect the presence of coronary atherosclerosis.

Medical Management of CHD

Over the past two decades, remarkable advances have been made in the management of angina pectoris. Pharmacologically, three major classes of drugs have radically changed the medical treatment of angina (Kloster & Bristow, 1985). These are: (a) the nitroglycerin (NTG) preparations and related nitrates, (b) the beta-adrenergic blocking drugs, and (c) the calcium entry blocking drugs. These drugs, alone or in combination, are reported to be

highly effective in preventing or relieving chest pain in CHD patients. There is some evidence to suggest that they may reduce CHD mortality and other complications of coronary atherosclerosis (Kloster et al., 1985). Each of these drugs is described briefly in the following section. Unless otherwise noted, the majority of the following information is taken from two sources— Kloster et al (1985) and McCall, Walsh, Frolich, & O'Rourke (1985).

Nitrates. The use of nitrates was introduced 100 years ago as antianginal agents. Today they remain the mainstay for treatment of angina. These drugs owe much success to their rapid onset of action with subsequent prompt and complete relief of pain. A number of preparations are available including oral, transdermal, and sublingual forms. Although the physiological consequences are not completely understood, it appears that the main sites of action are the specific receptor sites in vessel walls that produce dilation of vascular and other smooth muscle throughout the body. The most potent effects are on the venous system, with lesser effects on the large arteries. The effects of nitrates on the peripheral veins, the systemic arteries, and the coronary circulation, contribute to the beneficial responses of decreased myocardial oxygen demand and improved myocardial perfusion.

Beta-adrenoreceptor blocking drugs. Beta-adrenoreceptor blocking drugs are among the most widely prescribed medications. These drugs, also known as beta antagonists, are competitive inhibitors of catecholamine binding at beta-adrenergic sites,

thereby blocking or decreasing the effects of catecholamines. The mechanisms of action and the efficacy of these drugs are more clearly understood than is the case for nitrates. According to their sites of action, two types of beta-blockers have been identified. Beta-1 drugs block cardiostimulation and lipolysis. Beta-2 blockers block vasodilation. The cardiac effects of beta-1 antagonists result in decreased heart rate and decreased myocardial contractility as do all beta blockers. They do differ in terms of their non-cardiac effects on beta-2 receptors in the peripheral circulation and in the bronchi. This selectivity, though, is relative because at higher doses, both receptors are blocked (Durel, Krantz, Eisold, & Lazar, 1985). Although beta-antagonists do vary in terms of potency, this is clinically unimportant because dosages are titrated. By decreasing heart rate and contractility, beta blockers reduce myocardial oxygen demand, and decrease systemic arterial blood pressure and cardiac output. Systemic vascular resistance is increased. Beta-antagonists also differ, aside from their cardioselectivity, in other ways as well. Some (e.g., propranolol) have a quinidine-like effect making them useful in the treatment of arrhythmias. Beta-antagonists differ with respect to solubility with some being lipid soluble and some being water soluble.

Although differences do exist, there does not appear to be any particular advantages or disadvantages associated with any of them. They have been reported to be equally effective, resulting in symptomatic relief in 80 per cent of patients. Another drug

may be chosen, however, when certain contraindications exist (e.g., congestive heart failure, obstructive airway disease, or diabetes) or when potentially harmful side effects are anticipated (e.g., bradycardia). They are often used in combinations with nitrates or calcium entry blockers.

Calcium entry blockers. These drugs represent a heterogeneous group sharing in common the ability to inhibit the movement of calcium ions across myocardial and vascular smooth muscle. Three are currently approved for use in the United States, nifedipine, verapamil, and diltiazem, for the management of chronic stable angina in patients intolerant (secondary to side effects or contraindications) or refractory to treatment with beta-blockers and nitrates. Calcium entry blockers are also used to manage coronary artery spasm (a variant of angina). They are reported to be at least as effective as beta-antagonists and nitrates. The mechanism of action appears to be a decrease of or inhibition of calcium into the interior of the cell, and thus they modify a number of calcium-dependent processes (e.g., excitation-contraction in vascular smooth muscle). The direct cardiovascular effects are qualitatively similar resulting in decreased force of contraction, dilation of the coronary and systemic arteries, and slowed heart rate. All three also reduce systemic arterial pressure and vascular resistance, thus decreasing myocardial oxygen demand.

The Type A Behavior Pattern

The role of psychosocial factors in cardiovascular disease

was first empirically studied by two cardiologists, Friedman and Rosenman. Their research, which began in the 1950s, resulted in the formulation of the Type A behavior pattern (TABP). The TABP is best characterized as a set of overt behaviors which can be elicited from susceptible individuals given appropriately challenging environments (Matthews, 1982). The central elements of the behavior pattern appear to be a sense of time urgency, easily aroused hostility, and competitive achievement striving. Type A, which has been termed the "new" risk factor for CHD (Price, 1982), has been demonstrated in several epidemiological studies to be independently associated with an increased incidence of heart disease (Rosenman et al., 1964; Haynes, Levine, Scotch, Feinleib, & Kannel, 1978). The findings of such research, together with the emergence of the field of behavioral medicine and the need for a preventive approach to CHD, has resulted in a recently renewed interest in Type A behavior. This is reflected, in part, by the fact that, of all Type A studies published since 1959, greater than half have appeared in the last five years (Price, 1982).

Epidemiological Studies

As mentioned, the work of cardiologists, Friedman and Rosenman (1959), set the stage for empirical evaluation of the role of stress in cardiovascular disease. Frustrated by the inability of traditional risk factors, or combinations of these risk factors, to predict more than half of the new cases of CVD, these cardiologists began to observe their cardiac patients for

common emotional characteristics. Over twenty years of study resulted in the following definition of Type A Behavior:

"A characteristic action-emotion complex which is exhibited by those individuals who are engaged in a relatively chronic struggle to obtain an unlimited number of poorly-defined things from the environment in the shortest period of time, and, if necessary, against the opposing efforts of other things or persons in the same environment" (Friedman & Roserman, 1974, p. 67).

Persons without this style of behavior, who instead maintain a more relaxed, unhurried lifestyle, are described as Type B individuals. The Type B behavior pattern is often thought of as being the antithesis of Type A. This is probably not an accurate characterization because the Type B individual may possess some Type A behaviors, but not to the exaggerated degree that a Type A will show them. Matthews (1982) notes that the exact constituents of Type B behavior remains a matter of controversy. In her review of the Type A literature, she concludes that Type A is not a trait or a discrete typology but is instead thought to occur on a continuum ranging from extreme Type A to extreme Type B.

Friedman and Roserman (1959) characterized Type A by the following: "1) an intense, sustained drive to achieve self-selected but usually poorly defined goals, 2) a profound inclination and eagerness to compete, 3) a persistent drive for recognition and advancement, 4) a continuous involvement in

multiple and diverse functions constantly subject to time restrictions (deadlines), 5) a habitual propensity to accelerate the rate of execution of many physical and mental functions, and 6) an extraordinary mental and physical alertness" (p. 1286).

The initial research efforts of Friedman and Rosenman began with investigations of possible biochemical correlates that might accompany the TABP. Friedman, Rosenman, and Carroll (1958) studied 40 male accountants before, during, and after periods of increased occupational stress. Type A behavior was assessed using a personal interview which later became the Structured Interview (Friedman et al., 1964). Their results indicated that exposure to a stressful situation led to a marked rise in serum cholesterol and an acceleration of blood coagulation that was independent of diet and physical activity.

Friedman et al. (1959) randomly selected three groups of men, reported to differ only with respect to behavior pattern, from various occupational levels to further study the results previously found with the accountants. Three groups were formed, Type A, B, and C, and compared with respect to serum cholesterol, blood clotting time, presence of clinical coronary disease, and presence of arcus senilis. Arcus senilis is a term referring to the presence of a gray opaque ring surrounding the margin of the cornea and resulting from lipid degeneration (Dorland, 1982). The Type C group was similar in behavior to the Type B group but Group C also showed chronic anxiety. Results suggested an overwhelming difference between the groups of men with respect to

incidence of heart disease. Men in group A (fully developed Type A) were found to be seven times more likely to have clinical artery disease than men from Groups B or C. Group A was also reported to show significantly higher serum cholesterol levels, faster clotting times, and three times the incidence of arcus senilis. These authors reported that these results were not attributable to differences in exercise, fat intake, alcohol, or cigarettes. They concluded that it was likely that the behavior pattern accounted for the differences in the dependent variables.

In a now classic prospective study, the Western Collaborative Group Study (WCGS), Roserman, Friedman, Straus, Wurm, Kositchek, Hahn, and Werthessen (1964, 1975) examined the predictive relationship between TABP and CHD. These authors hypothesized that if the behavior pattern plays a significant role in accelerating CHD, then healthy men, without CHD, with this behavior pattern, should show a higher future incidence of CHD. Subjects were 3,154 employed males, aged 39 to 59 years, recruited from ten business organizations in California. Overt behavior pattern was assessed using a personal interview, the Structured Interview. This interview contained 27 items requiring subjective interpretation by the examiner. There were six content areas included in the interview: (a) history of past social achievements, (b) present drive to achieve, (c) competitive involvement, (d) time urgency, (e) hostility, and (f) overt stylistic signs of TABP (e.g., appearance of urgency, gesturing, explosive speech). TABP was observed in 1,589 of the men, and

Type B behavior was determined in 1,565 of the men. A thorough history (e.g., history of illness, family history, education, occupation, physical activity, diet, alcohol, and tobacco use, etc.) was obtained and dependent variables—serum lipid and lipoprotein studies, blood coagulation studies, and cardiovascular examination—were collected at intake and annually for eight to nine years. Results at follow up indicated that Type As were twice as likely to develop CHD than Type B subjects. In addition, Type As, as compared to Bs, were found to have: (a) five times the frequency of suffering a second myocardial infarction (MI), (b) twice the frequency of fatal MIs, (c) twice the degree of coronary atherosclerosis at post mortem examination in the 25 patients who died. Eighty-eight per cent, or 22 of 25, of the subjects who died of CHD were assessed at intake as Type A. Rosenman et al. (1975) reported that this predictive association between Type A and CHD was not attributable to traditional risk factors. These authors suggested that the TABP should be considered prominent among the major risk factors for CVD. They emphasized that Type A was not an artifact of other risk factors and that the pathogenic force of the behavior pattern was not due to other risk factors. They speculated that the danger associated with Type A behavior might operate through neurohumoral mechanisms.

Twenty-five of the subjects from the WCGS, were later found to show that evidence of a "silent MI" (by electrocardiography) that was unknown to the subjects or the WCGS investigators at the

time of data collection. Jenkins (1966), with the cooperation of Friedman and Roseman, designed a study intended to objectify the components of TABP and to examine the behavioral and biochemical attributes of these men identified as having suffered from "silent MIs". Two control groups were used: (a) a group matched for age and occupation, and (b) a group matched on age, occupation, and behavior (A or B). Both control groups were found to be free of any signs of CHD. As in the original WCGS, the scores of the six behavior components and biochemical data were analyzed. Regarding behavior type, it was found that the scores from the "silent infarct" subjects were significantly higher than the control group scores on record of past achievements, manifest hostility, and total score. Further, the men in the "silent infarct" group who scored high on impatience and time urgency also had a history of higher achievement with regard to job status. With respect to blood lipid measures, it was reported that subjects with high levels of hostility showed significantly higher mean serum beta lipoprotein and beta-alpha lipoprotein ratios. High scores on the achievement component were associated with higher total serum cholesterol levels. This data led Jenkins (1966) to suggest that, perhaps, the TABP was not a unidimensional construct, but instead that various combinations of traits were associated with various biochemical variables. The work of Jenkins (1966) lends support to the findings of the WCGS and also emphasizes the need for a more objective approach.

Another classic epidemiologic study in the area of Type A

research was conducted by Haynes et al. (1978, 1980) with the Framingham Heart Study. Using 1822 men and women of the Framingham cohort, aged 45-77, these authors investigated the role of psychosocial stress in the etiology of blood pressure, serum cholesterol, and smoking. Subjects were free of clinical symptoms of CHD at the initiation of the study. They were followed annually for a period of eight years. In a three part series of publications resulting from this study, Haynes et al. (1978a, 1978b, 1980) describe the development of the Framingham Type A Psychosocial Interview and report correlational data on a variety of psychosocial factors and CHD, including the association between psychosocial stress and CHD risk at eight year follow-up. The interview developed by these authors included 20 scales that can be grouped into four main categories: (a) Behavior types—including Framingham Type A Behavior, emotional lability, ambitiousness, and non-easy going scales, (b) Situational Stress—including scales measuring situations from job, marriage, and life, (c) Somatic strains—including measures of tension, daily stress, anxiety, and anger, and (d) Sociocultural mobility including scales measuring occupational changes, promotions, educational, and job mobility.

Results revealed that the Framingham Type A Behavior Scale was correlated with ambitiousness, emotional lability, tension, daily stress, anger symptoms, educational level, and occupational status. Sex differences were observed, with women being less likely to exhibit the Framingham Type A behavior but showing more

signs of emotional lability, anger, tension, and anxiety. Less consistent differences were found between measures of psychosocial stress and blood pressure or cholesterol. There was no association found between scores on the Framingham Type A Behavior Scale and standard CHD risk factors.

At follow-up (eighth or ninth biennial exam), the data revealed that subjects with all four categories of CHD (i.e., uncomplicated angina, angina with or without MI, MI, and total CHD) scored higher than did subjects without CHD manifestations. In a multivariate analysis, the Framingham Type A Behavior Scale differentiated between men with and without MI even when traditional risk factors were controlled. The degree of coronary risk associated with the Type A behavior for men 39-59 was 2.2 for total CHD, 2.1 for MI, and 2.5 for angina pectoris only. Coronary risk for men aged 45-64 was found to be lower: 1.8 for total CHD, 2.1 for MI, and 1.8 for angina. It is noteworthy that this association was significant only among men having white-collar jobs. The differences between Type A and Type B disappeared when blue-collar workers were studied. When other psychosocial stress scales were included in the analysis, a scale reflecting aging worries was found to be the only significant discriminator. Among the women, both the Framingham Type A Behavior Scale and the emotional lability scales were correlated with CHD prevalence when other risk factors were controlled. The coronary risk associated with Type A behavior in women (under 65) was found to be 2.1. Further, suppressed hostility was found to be an independent

predictor of CHD among white-collar men and working women.

In conclusion, the work of Haynes et al. (1978a, 1978b, 1980) supports previous data indicating that Type A behavior is associated with increased coronary risk, despite the use in this study of an alternative method for assessing Type A. However, in the Framingham study, only white-collar men and employed women were found to have a significant association between TABP and increased coronary risk. The importance of hostility as a contributing factor was also demonstrated in this study.

Brand, Roserman, Sholtz, and Friedman (1976) compared the results of the WOGS and the Framingham study using a multiple logistic method of analysis. After adjusting for length of follow-up, these authors concluded that the CHD risk predictions from both studies demonstrated good agreement. They found that the relative risk of CHD (obtained from estimated logistic coefficients for behavior pattern) was 1.90 and 2.10 for younger and older decades, respectively. That is, after traditional risk factors were adjusted, younger Type A men (aged 39-49) had 1.90 times the risk for CHD than Type B men. Older men (aged 50-59) showed 2.10 times the risk of CHD when compared to Type B men in the same age category. Brand et al. (1976) also investigated what CHD reduction would occur if the direct risk associated with TABP was eliminated. They found that for the younger group, a 28.5 per cent risk reduction would occur. A 32.3 per cent reduction was reported for the older group. Combining these groups resulted in a 31 per cent risk reduction rate.

Disconfirming results on the association between CHD and Type A behavior have been published. One recent study (Case, Heller, Case, & Moss, 1985) reported that Type A (assessed using the JAS) was not related to cardiac mortality or other disease indices (e.g., left ventricular ejection fraction) in 516 post-infarct subjects. Given this data, some researchers have argued that the JAS does not accurately assess Type A. However, other sources of error are equally likely. Previous research (Roserman et al., 1964) has indicated that Type A individuals are more at risk of dying from heart attacks. It is possible, then, that subject mortality resulted in a biased sample in this study. Secondly, it is conceivable that the experience of having a heart attack might lead some patients to change their behavior. At any rate, negative findings from one study, that stand in direct contrast to a substantial number of studies demonstrating positive findings, do not automatically negate the association between Type A and CHD. Instead, the findings of Case et al. (1985) should serve as an impetus for continued research.

In 1981, the results of the Review Panel on Coronary-Prone Behavior and Coronary Heart Disease, sponsored by the National Heart, Blood, and Lung Institute, were published. This panel consisted of biomedical and behavioral scientists whose task was to critically evaluate the existing scientific knowledge on the proposed association between behavior and CHD. After an extensive review of the literature, these panel members concluded:

"The review panel accepts the available body of

scientific evidence as demonstrating that type A behavior—as defined by the structured interview..., the Jenkins Activity Survey ..., and the Framingham type A behavior scale—is associated with an increased risk of clinically apparent CHD in employed, middle-aged U.S. citizens. This risk is greater than that imposed by age, elevated values of systolic blood pressure and serum cholesterol, and smoking, and appears to be of the same order of magnitude as the relative risk associated with the latter three of these factors" (p. 1200).

Assessment of Type A Behavior

The two most commonly used methods for assessing Type A behavior are the Structured Interview (SI, Roserman et al., 1964) and the Jenkins Activity Survey (JAS, Jenkins, Roserman, & Friedman, 1967). Both of these measures have been shown in numerous studies to discriminate CHD subjects from non-CHD subjects and to predict future incidence of CHD in subjects who were initially well. The Framingham Type A Behavior Scale (Haynes et al., 1978, 1980) has also been found to have these properties, but aside from the Framingham Heart Study, it has not been widely used. Other Type A measures have also been developed (e.g., Bortner Rating Scale, Bortner, 1969) but, as yet, these measures have not been shown to have a predictive association with CHD.

Structured Interview . The SI (Roserman et al., 1964) is a 25 item provocative interview situation in which Type A assessment

is based on the voice stylistics, psychomotor behaviors, and verbal content of the respondent (Rosenman, 1978). It was developed at the Harold Brunn Institute in San Francisco, California for use in the Western Collaborative Group Study (WCGS). It is best characterized as a challenge situation in which a trained interviewer/observer attempts to bring out covert Type A behaviors (Rosenman, 1978). Some of the questions are dependent solely on verbal content of the response (e.g., "Do you think you drive harder to accomplish things than most of your associates?"). Other questions are asked in such a way as to elicit Type A behavior. For example, the interviewer asks the subject a question in a deliberately slow, hesitant fashion, anticipating the Type A subject to become annoyed and/or interrupt the interviewer. Subjective assessment is then made based on the presence of observed motor and verbal stylistics thought to characterize the Type A individual (e.g., rapid, loud, emphatic speech; frequent use of gestures; frequent interruptions of the interviewer). Four assessment categories are used: (a) Fully developed Type A (A-1), (b) Incompletely developed Type A (A-2), (c) Absence of Type A (Type B), and (d) Type X in which categorization is not possible secondary to an equal representation of both Type A and Type B.

An 80 per cent test-retest agreement rate has been reported for the SI. Interrater reliability estimates range from .75 to .90 (Rosenman, 1978). The validity of the SI has been well documented as subsequent sections of this paper will

illustrate.

Jenkins Activity Survey The SI, although shown to be reliable and valid, is not necessarily the ideal method for the assessment of Type A Behavior. It is not truly objective and does not allow for numerical quantification (Rosenman, 1978). It requires specialized training, supervised experience, and periodic quality control in the form of monitoring recorded interviews so that observer bias can be prevented (Jenkins, 1978). Some authors have noted that it may have limited applicability to some practitioners because of these factors and the fact that findings may be largely dependent on the interviewers' skill (Jenkins, Friedman, & Rosenman, 1967). The Jenkins Activity Survey (JAS, Jenkins et al., 1967) was developed to duplicate the SI psychometrically with the aim of providing a more standard, objective approach to the assessment of Type A behavior.

The JAS (Form C) is a self-report, machine-scorable questionnaire. It uses a multiple choice response format requiring the respondent to choose which response is most true. An Overall Type A score is derived along with three factor scores—Speed and Impatience (Factor S), Job Involvement (Factor J), and Hard-Driving and Competitive (Factor H).

As mentioned, the JAS was developed to duplicate the SI. Jenkins worked closely with Friedman and Rosenman learning the SI and observing many SI assessments. From this, he developed an original item pool of 64 questions. Instead of relying on the face validity of the 64 original items, the JAS was subjected to a

series of empirical analyses using subjects and data originally collected for the WCGS. In 1964, the experimental version of the JAS was administered to 120 men in the WCGS whose behavior pattern had been originally assessed using the SI. Forty items of the experimental JAS significantly discriminated between SI-determined Type As and Type Bs. These 40 items, along with an additional 21 items, comprised the first edition of the JAS (Jenkins, Rosenman, & Zyzanski, 1965). This version was empirically validated using 2,951 men of the WCGS assessed by the SI as Type A or Type B at test (1960) and retest two years later. These men were divided into three groups: (a) 400 Type As and 307 Type Bs, (b) 475 Type As and 509 Type Bs, and (c) an independent sample of 409 men. The responses of Group 1 were used to scale items and weight response alternatives so that weighted scores could be treated as a continuous variable on an approximately equal interval scale and so that there would be a maximum distinction between criterion groups. Group 2 served as a cross-validation sample using weighted response items which significantly discriminated between As and Bs in group 1. A discriminant function analysis (using 31, 28, 24, and 19 items) was applied to the items surviving this cross-validation in order to compare for item sensitivity and specificity with respect to the identification of Type A behavior. The 19 item equation was found to be the best predictor of Type A behavior. These 19 items were retained and cross-validated using group 3.

An approximately normal distribution was found with the

scores of all 2,951 men tested. Scores were transformed to yield a mean of 0.00 and a standard deviation of 10.0. Subjects scoring greater than or less than one standard deviation from the mean were found to be correctly classified as Type A or Type B by both the SI and the JAS. The intermediate scores were found to be less accurate for predicting Type A behavior. The level of agreement between the 1965 JAS and the 1960 and 1962 SI was determined using the third group. A 73 per cent agreement rate was found. Using only extreme scores (i.e., greater than or less than one standard deviation from the mean) resulted in a 90 per cent agreement rate (Jenkins, Zyzanski, & Roserman, 1971). Jenkins et al. (1979) reported these to reflect respectable levels of agreement considering the change in mode of data collection and the fact that three years had elapsed since the most recent SI.

A second edition of the JAS was published in 1966. Redundant items or items with less than adequate psychometric properties were dropped for this edition and several new items were added. This 57-item version was then validated and cross-validated in much the same way as was done with the 1965 version. Twenty-six items were found to discriminate between Type A and Type B men. Some of these items were the same as were found with the 1965 version, although a number of new items emerged. Cross-validation procedures revealed a 71 per cent agreement rate between the 1966 JAS and the 1960 and 1962 SI.

In order to address the question of whether Type A behavior was a single syndrome or an aggregation of subsyndromes, a series

of factor analytic procedures were conducted. Using the data obtained from the 1965 and 1966 JAS validation studies, three factors were identified (Zyzanski & Jenkins, 1970). These were labeled as Factor S (Speed and Impatience), Factor J (Job Involvement), and Factor H (Hard-Driving and Competitive). Jenkins et al. (1979) described the three factors in the following ways. Factor S refers to a perceived sense of time urgency. Subjects scoring high on this factor tend to be impatient with others, are likely to hurry others along, and are prone to irritability. Subjects scoring high on Factor J tend to show high levels of dedication to their job. They are likely to work longer hours, to take work home with them, and report often being confronted with deadlines on the job. High scores on Factor H are found in individuals who perceive themselves as being serious, competitive, and hard-working, often feeling as though they put in much more effort than their colleagues.

Another version of the JAS was published in 1969. On this occasion, the authors felt that the SI from nine years previous would be unsuitable to use as validation criteria. Instead, the 1965 and 1966 editions of the JAS were used. A discriminant function analysis was conducted to ascertain the items making the strongest contribution to each of the four scales.

A fourth revision (Form B) was then published in 1972 which contained those items shown to discriminate between As and Bs in the 1969 validation and two new items. Items containing gender references were also reworded, making the JAS equally applicable

to females as well as males.

The most recent version, Form C, was used in the present study. The two experimental items added in Form B were not included in Form C as they did not load on any of the four JAS scales. Form C, then, contains 52 items. The item composition and scoring algorithms in Form C are exactly the same as those in the 1969 edition and Form B. Therefore, all psychometric data reported in the JAS Manual (Jenkins et al., 1979) for the 1969 edition and Form B apply equally to Form C.

Jenkins et al. (1979) provides normative data based on the 1969 JAS scores of 2,588 males employed in middle- and upper-echelon jobs who served as subjects in the WCGS. Norms on populations other than this (predominantly males) are also available.

Measures of reliability of the JAS have been reported. Test-retest correlation coefficients, using intervals of one to four years, were found to range from 0.60 to 0.70. These correlations were found despite modifications in the successive editions of the JAS, suggesting that the traits measured by this instrument are relatively stable for at least up to a four year period. Internal consistency, the degree to which items in a scale measure a unified concept, has also been computed. These reliability coefficients for all four JAS scales range from 0.73 to 0.85.

The validity of the JAS has been well documented. Subsequent sections of this paper will report findings indicating that the

JAS predicts CHD incidence and severity of atherosclerosis. As has been previously mentioned, rates of agreement between the JAS and the SI range from 73 to 90 per cent, depending on whether extreme classifications are used.

Type A and Stress

In light of a growing wealth of evidence indicating the significant role of Type A behavior in CHD, many researchers have turned their attention toward investigations aimed at assessing the possible underlying physiologic mechanisms. Research, thus far, has suggested that physiologic responsiveness (i.e., reactivity) to emotional stress may be a marker of correlated pathogenic processes in CHD or might serve as direct contributors to CHD pathology (Krantz & Manuck, 1982). Although many individual difference variables are involved in the cardiovascular and endocrine response to stress (Krantz et al., 1982), research by Eliot (1982) and others suggests that some individuals show exaggerated cardiovascular responses to challenge situations that may make them coronary-prone. Results from numerous human and animal studies (Manuck, Kaplan, & Clarkson, 1983) support this notion, and suggest that pathogenic states can be evoked, even with short term exposure to psychological stressors (Krantz et al., 1982).

Reactivity involves the measurement of changes from baseline functioning that result from exposure to a laboratory or naturalistic challenge. If acute changes, produced by psychologic stimuli, can be linked to potentially pathogenic states, then

these responses may serve as more useful predictors than traditional risk factors that are usually obtained at rest (Krantz et al., 1982).

Atherosclerosis

Before continuing with a thorough discussion of reactivity, it may be useful to briefly review the atherosclerotic process in which most of the coronary artery lesions that result in CHD can be classified (Wissler, 1985). Atherosclerosis can be defined as a form of arteriosclerosis (i.e., thickening in the walls of the arterioles) in which lipid filled atheromas (i.e. plaques) form within the intima and inner media of large and medium sized arteries (Dorland, 1982). Of uppermost concern here are those plaques that form within the walls of the coronary arteries. It is beyond the scope of the present paper to review the various etiologies proposed for atherosclerosis. The interested reader is referred to Wissler (1985). Suffice it to say that one of the ways that these changes can be brought about is by hemodynamic or chemical injury (Eliot, 1982). Hemodynamic injury results from impaired blood flow or turbulence at the site of the lesion. Chemically, it is also known that certain blood lipids (e.g. low density lipoproteins as well as cortisol and catecholamines) at certain levels are toxic to coronary arteries, and thus, can accelerate the atherosclerotic process and/or prevent normal healing (Eliot, 1982). Further, plaques can continue to grow in size secondary to continued lipid and cholesterol deposits, as well as from the entrance of blood platelets which are released.

The end result can be arterial stenosis (which can further restrict blood flow), calcification, arterial spasms (responsible for some angina attacks), or worse, ulceration and thrombus (i.e., clot) formation. It is the latter event, the development of a coronary thrombus, that usually leads to a classical myocardial infarction (Wissler, 1982).

There is evidence to suggest that Type A persons show elevated blood pressure, heart rate, catecholamines, ACTH, cortisol, and serum cholesterol when confronted with appropriate challenges (Krantz et al., 1982). Thus, theories relating to the sympathetic-adrenal-medullary and pituitary-adrenal-cortical systems are the pathways through which behaviorally mediated risk mechanisms may operate (Eliot, 1982). The catecholamine surges resulting from sympathetic nervous system activation can have as consequences the promotion of platelet adhesiveness and aggregation, arrhythmias, as well as the secretion of a number of hormones, and a decrease in insulin secretion (Eliot, 1982). Glucocorticoids, released via the pituitary-adrenal-cortical axis, promote the development of diabetes, hyperlipidemia, and hypercholesteremia. Glucocorticoids enhance water diuresis, decrease circulating lymphocytes, increase platelet counts (which in turn enhances clotting tendencies), and increases gastric acidity and pepsin production. They also block growth hormone secretion, decrease calcium absorption, increase angiotensin production, suppress inflammatory responses, lower the electrical excitation threshold of the brain, and sensitize arterioles to the

pressor effects of catecholamines (Eliot, 1982). It should be noted that, although these systems do appear to affect CHD, the precise mechanisms through which they operate are still a matter of speculation (Krantz et al., 1982). This list of potentially adverse effects lends further credence to the importance of examining the mechanisms of reported reactivity in the Type A individual.

Stress

As mentioned, the two systems thought to be related to reactivity are the sympathetic-adrenal-medullary system and the pituitary-adrenal-cortical system. A more detailed analysis of how these systems are related to stress is necessary. As originally defined by Selye in 1936, the term stress in the present paper will refer to the "nonspecific (that is, common) result of any demand on the body, be it mental arithmetic, somatic survival, and the accomplishment of our aims" (Selye, 1980, p. vii). During the course of his research, Selye modified the definition to more clearly distinguish between pleasant and unpleasant stress. He notes, however, that the distinction proves to be immaterial since it is the intensity of the demand for readjustment or adaptation that determines the response (Selye, 1974). The use of the term nonspecific does not imply that all stress is identical. The stressor, that agent which produces stress, will necessarily have its specific effects. This specificity of the stressor does not contradict the term nonspecificity contained in the definition of stress. Instead,

the specificity of the stressor can be explained by the stressors side effects and the internal and external predisposing factors that may modify the response.

The definition of stress proposed by Selye is based on his early research showing that there were three objective indicators that could be recognized no matter how stress was produced. These indicators were (a) the mobilization of the anterior pituitary-adrenal axis, (b) the reliable involution of the thymico-lymphatic system, (c) the appearance of peptic ulcers. The appearance of these events, as well as other nonspecific indicators, defines the phenomenon of stress, irrespective of other changes that may occur following exposure to the stressor (Selye, 1980, p.vii). It would be misleading, though, to consider stress as a dichotomous yes-no type of phenomenon. Stress, according to Selye, exists in varying degrees. That is, different demands of different intensities may not always result in identical or similar reactions. In each situation, the role of stress may be more or less important.

One source of confusion in the stress research involves the use of different formulations to conceptualize stress (McNamara, 1982). A frequently used conceptualization is that of stress as an environmental event. This approach is most consistent with the study of the association between the accumulation of life events and the risk of physical illness. Holmes and Rahe (1968) stimulated research in this area by reporting strong positive correlations between exposure to a large number of life changes

and the risk of developing some kind of illness. It is beyond the scope of the present paper to review the tremendous proliferation of research that followed such findings. Suffice it to say, that such research was strongly criticized for yielding unreliable scores and including items reflecting illness (Holroyd, 1979). Rabkin and Struening (1976), after an extensive review of the literature, concluded that life events have not been shown to be reliable predictors of the probability of future illness. Life events research, while drawing attention to important sources of stress, tends to focus on linear associations between independent and dependent variables. Rabkin et al. (1976) call for future research to focus on the assessment of internal mechanisms through which life events may have their impact.

Another well known approach to stress is that of stress as a response. This area is best exemplified by the work of Selye and his description of the General Adaptation Syndrome (GAS, Selye, 1936). The GAS (Selye, 1936) is a nonspecific response of the body to an intense or noxious demand. There are three distinct phases of the GAS--alarm, resistance, and exhaustion. Selye (1974) has proposed that prolonged or repeated elicitation of the GAS, or defects associated with this response, play an important role in a large number of stress-related diseases, including cardiovascular disease. The changes associated with these phases are mediated by the release of adrenocorticotrophic hormone (ACTH) which stimulates the release of glucocorticoids, and the activation of the autonomic nervous system which liberates

catecholamines from the sympathetic nervous system and by the adrenal medulla (Selye, 1974). Thus, "the body responds to increased physical or psychological demands by releasing adrenocorticotropin (ACTH) from the anterior pituitary, glucocorticoids from the adrenal cortex, epinephrine from the adrenal medulla, and norepinephrine from the sympathetic nerves" (Axelrod & Reisine, 1984, p. 452).

Although the work of Selye has provided an eloquent documentation of the pituitary-adrenal axis in response to a number of demands, it falls short of providing information about the role that psychological variables may play in influencing this response. Mason (1968, 1971) has argued that the stress response is mediated by psychological variables. A third approach to the study of stress is the transactional model. In this approach, stress is conceptualized as a transaction between a system and its environment. Stress is characterized by the cognitive processes that mediate the adaptive demands to the individual and the individuals' response to them. Lazarus (1966, 1971), using this approach, conceptualizes stress as an interaction between appraisal and coping processes.

Space limitations do not permit an extensive review of each of the factors involved in the stress response. The interested reader is referred to Axelrod et al. (1984) and Mason (1968). The focus of the following discussion will be on ACTH, because it is the hormone measured in the present study. When pertinent, however, interactions with other substances will be described.

Adrenocorticotrophic Hormone

The release of ACTH is thought to be the main indicator of the onset of the "stress syndrome" or GAS (Makara, Palkovits, & Szentangothai, 1980). The influence of psychological factors on the pituitary-adrenal-cortical system has been, perhaps, the most extensively studied of the endocrine systems, owing much to the work of Selye and to the availability of recently developed excellent biochemical methods of measurement (Mason, 1968). Human ACTH is a 39 amino acid, anterior pituitary polypeptide hormone (Krieger, 1979) controlled by complex regulatory mechanisms such as corticotropin releasing factor (CRF), vasopressin, catecholamines, and possibly other as yet unknown hormones (Axelrod et al., 1984). Several of these deserve brief elaboration. It is noteworthy that the release of ACTH in animals by catecholamines at sufficiently high levels (eg., that occurring during hypoglycemia and with myocardial infarction) can be blocked by propranolol, a beta-adrenoreceptor blocking drug. Further, isoproterenol-induced ACTH secretion has been reported to be blocked by calcium antagonists or a lack of extracellular calcium (Reisine, Heisler, & Hook, 1982).

Once secreted, ACTH stimulates synthesis of cortisol (in humans) which, in turn, serves to inhibit further secretion of ACTH (Axelrod et al., 1984). Because alternative methods of ACTH regulation have been reported, the measurement of changes in corticosterone (or cortisol) and adrenal weight, the most frequently used measures of stress, cannot be assumed to reflect

pituitary ACTH in a quantitative manner (Krieger, 1979). In addition, the measurement of ACTH is thought to be at least one step closer than corticosterone to the site of neural initiation of the stress response (Vernikos-Danellis & Heybach, 1980).

The response of ACTH to an emotional stimulus is influenced by both the quality and strength of the stimulus (DeWied, 1980). Although the pituitary-adrenal system responds to a wide variety of psychological stimuli, those with an element of novelty, uncertainty, and unpredictability (Mason, 1968) or those resulting in fear, anxiety, and frustration (Selye, 1950), are among the most potent stressors for system activation, resulting in reportedly striking responses. The influence of stress upon ACTH has been said to be superimposed upon all other regulators of ACTH. Regardless of time of day or level of plasma cortisol, the normal individual responds to major stress with an increase in ACTH and a consequent increase in cortisol (Daughaday, 1981).

It is critical for researchers in the area to note that there exists a circadian periodicity for ACTH secretion (Krieger, 1978). This rhythmicity occurs in episodic, relatively synchronous peaks, with the majority of upward peaks occurring between 3:00 a.m. and 9:00 a.m. so that the highest ACTH levels are found upon awakening. Wakening is followed by a downward trend with episodic peaking (associated with mealtimes) and quiescent periods (Krieger, 1979). The normal range for early morning samples is 21-139 picograms per milliliter (pg/ml) of blood plasma. Late evening samples range from 20 to 85 pg/ml (Ruhmann-Wennhold &

Nelson, 1979). ACTH periodicity is reported not to be affected by stress and is not dependent on corticosterone feedback (Krieger, 1979). There has been some controversy regarding whether ACTH rhythmicity is normal or abnormal in psychiatric patients. Krieger (1978) notes that there appears to now be general agreement that such periodicity is normal, having perhaps more frequent episodic release.

Reactivity Studies

Studies show, in general, that Type As, as compared to Bs, respond to appropriate challenges, be they laboratory induced or naturalistic, with increases in blood pressure, heart rate, catecholamines, ACTH, cortisol, and cholesterol (Krantz et al, 1982). Hemodynamic and endocrine findings have led researchers to speculate about the roles of the sympathetic-adrenal-medullary and pituitary-adrenal-cortical stress response systems. The purpose of the present discussion is to relate the results of numerous studies that have accumulated in this area.

How and why the Type A behavior pattern confers coronary risk can be studied in several ways. First, do Type As behave in such a way as to frequently arouse the sympathetic nervous system? The greater bulk of the evidence here points to the importance of the roles of hostility, aggressiveness, and competitiveness in Type A (Matthews, Glass, Rosenman, & Bortner, 1977; Carver & Glass, 1978; Williams, Haney, Lee, Kong, Blumenthal, & Whalen, 1980; Gentry Chesney, Gary, Hall, & Harburg, 1982; Barefoot, Dahlstrom, & Williams, 1983; Shekelle, Gale, Ostfeld, & Paul, 1983). These

investigations, which assess the role of hostility in Type A behavior, appear to follow two major directions. First, there are those studies that have demonstrated the existence of a predictive association between hostility in initially well subjects and subsequent development of CHD (Matthews et al., 1977; Barefoot et al., 1983; Shekelle et al., 1983). Similarly, Williams et al. (1980) and Dembroski, MacDougall, Williams, Haney, & Blumenthal (1985) reported significant positive associations between hostility and degree of coronary atherosclerosis that was independent of other factors.

A second way that the role of hostility has been studied is by subjecting Type As and Bs, primarily college students, to particularly difficult and frustrating tasks under conditions of harassment and non-harassment by a confederate. These studies (Glass, 1982; Carver & Glass, 1978) support hypotheses suggesting the critical role of hostility and aggressiveness in the Type A behavior pattern. Using a paradigm in which subjects were either exposed to a harassing or non-harassing confederate to whom they were to teach a complex perceptual motor task, Carver et al. (1978) found that Type As, assessed by the student form of the JAS, delivered significantly higher levels of shock intensity to the harassing learner than did Bs. The work of Glass (1982) supported these findings. Using a competitive game situation in which subjects were randomly assigned to harass and no-harass conditions, Glass (1982) reported significantly greater levels of systolic blood pressure, heart rate, and catecholamines (i.e.,

epinephrine) among As, as compared to Bs, that were attributable to condition. Glass (1982) concluded that, although competition alone does not result in significant differences between As and Bs, the response to an element of hostile interaction results in enhanced responding among Type A subjects. These studies provide evidence, that the role of hostility, with subsequent sympathetic nervous system activation, may be one of the ways that CHD confers coronary in Type A persons.

Another question that needs to be addressed when attempting to explain how Type A confers coronary risk, is whether there are some as yet unidentified components that lead to increased coronary risk among Type A individuals. The association between psychosocial variables (other than Type A) and CHD has been examined (Dimsdale, Hackett, Block, & Hutter, 1978; Jenkins, Stanton, Klein, Savageau, & Harken, 1983; Schweritz, McKelvain, Laman, Patterson, Dutton, Yusim, Lester, Kraft, Rochelle, & Leachman, 1983; Freeman, Fleece, Folks, Cohen-Cole, & Waldo, 1984). Results thus far have been contradictory. Some authors have reported significant positive correlations between Type A and stressful life events, tension, depressive mood, and anger (Dimsdale et al., 1978) as well as fatigue, life dissatisfactions, and sleep disturbance (Jenkins et al., 1983). Schweritz et al. (1983) failed to find significant associations between psychosocial measures and extent of disease. Finally, one study (Freeman et al., 1984) reported unexpected results. These authors found that the incidence of arrhythmias following coronary artery

bypass surgery was negatively correlated with measures of depression and anxiety. They reported that patients scoring low on depression and on anxiety had a greater incidence of postoperative arrhythmias. As can be seen, the results of these studies are far from conclusive and further research is needed before definitive statements can be made regarding the role of other psychosocial factors in CHD.

Before reviewing studies that examine whether Type As, as compared to Type Bs, demonstrate reactivity to stressful situations, an additional issue must be addressed. This concerns the question about whether there is a predictive association between reactivity and CHD development. Predictive associations have been reported showing that Type A Behavior is predictive of CHD development. There are also studies that show, in most cases, that Type A behavior is associated with degree of atherosclerosis (Blumenthal, Williams, Kong, Schanberg, & Thompson, 1978; Frank, Heller, Kornfeld, Sporn, & Weiss, 1978; Zyzanski, Jenkins, Ryan, Flessas, & Everist, 1976; Kahn, Kornfeld, Blood, Lynn, Heller, & Frank, 1982). Supportive data is also provided by Friedman, Roserman, Straus, Wurm, & Kositchek (1968). These authors examined the coronary arteries of 51 of the original WCGS (Roserman et al., 1964) sample who died after the initiation of the study. Twenty-five of these subjects died of CHD and the remaining 26 subjects died of causes unrelated to CHD. Twenty-two of the 25 CHD-related deaths (88 per cent) occurred in subjects assessed as Type A in the original WCGS. Further, these authors

found that severe atherosclerosis was six times more likely in Type A subjects. However, data regarding whether or not reactivity itself can predict CHD is lacking. This author was only able to identify one such study. In a 23 year prospective study, Keys, Taylor, Blackburn, Brozek, Anderson, & Simonson (1971) examined this issue. Subjects were 279 upper-class, employed men, aged 47-57, who were CHD-free at the initiation of study. These authors found that the most significant single predictor of CHD was diastolic blood pressure response to the cold pressor test. The hyperreactor group, compared to subjects not considered to be hyperreactors, was found to have 2.4 times the risk of CHD death or myocardial infarction. The next best predictor was a combination of cholesterol level and systolic blood pressure. Although the results of one study alone does have limited generalizability, the work of Keys et al. (1971) provides intriguing supportive data that reactivity is associated with disease development.

Returning to the issue at hand, that of how and why Type A confers coronary risk, one final group of data must be considered. The collection of studies that follow examined whether or not Type As, as compared to Type Bs, indeed show cardiovascular and endocrine reactivity when exposed to a stressor. If one is to accept the tentative hypothesis that reactivity is related to coronary risk, and that this reactivity operates via sympathetic adrenal medullary and pituitary-adrenal-cortical axes, then such reactivity must be demonstrated in a variety of subjects exposed

to a variety of stressful tasks. The purpose of the following discussion is to critically review these studies.

Numerous studies have been conducted which examine reactivity in cardiac patients manifesting the Type A behavior pattern. Aside from cardiac patients, several other populations have been used, including college students and healthy adults. Both laboratory-induced stressors (e.g., mental arithmetic, reaction time) and naturalistic settings/stressors (e.g., in the workplace, coronary artery bypass surgeries) have been used, although the use of the latter has been far more infrequent. Type A behavior has been assessed primarily with the SI and JAS in these studies. Finally, many different physiologic measures have been assessed. The diversity of these studies does result in some degree of confusion and controversy. The majority of the studies, however, demonstrate that Type A persons fail to differ from Type Bs under conditions of rest, but do tend to differ on some physiologic variables when exposed to appropriate stressors. Studies using laboratory-induced stressors will be considered first.

Using a reaction-time task, Dembroski, MacDougall, & Shields (1977) found that Type A college students, assessed by the SI, did not differ at rest but responded to the RT task with significantly greater increases in heart rate (HR) and systolic blood pressure (SBP) when compared to Type B students. Greater HR variability was also observed in the Type A group. No differences were found for galvanic skin potential. Jennings (1984) also examined reactivity using a RT task in healthy students assessed for

behavior type by both the SI and the JAS. SI-assessed Overall Type A was found to be significantly correlated with shorter vascular transmit times. Subjects scoring in the Type A direction of the Speed and Impatience component of the SI were also found to exhibit greater task-related HR changes. Subjects did not differ during baselines between trials. No significant associations were revealed for the JAS. It should be noted that a predictive association between Type A and CHD has not been shown for the student form of the JAS (Jenkins et al., 1978). The results of these studies suggest that Type As do not differ from Type Bs during periods of rest, there is increased responsiveness to periods of stress in Type A subjects. The work of VanEgeren (1979) also supports the finding of increased HR to stress among Type As. Using a game situation, this author found that healthy college undergraduates, males and females, assessed by the student form of the JAS, responded with significantly greater HR as compared to Bs. No significant differences were found for blood volume pulse or respiration. Again regarding HR, an interesting finding was reported by Hart and Jamieson (1983). They found that both Type As and Bs, assessed via the JAS-student form, responded to a perceptual conflict task with increases in HR. However, they noted a significantly slower HR recovery after the stressor in Type As, perhaps suggesting that maladaptively prolonged cardiovascular arousal might be involved in the mechanisms underlying Type A and reactivity.

Using cardiac patients and normal controls, Dembroski,

MacDougal & Shields (1977) found that SI-assessed Type A subjects showed greater increases in SBP and DBP, compared to controls on the SI and a stressful history quiz, despite the use of beta-blockers in the cardiac patients. Corse, Manuck, Cantwell, Girdani, & Matthews (1982) also made use of a difficult series of cognitive tasks to study reactivity. Healthy adult controls and cardiac patients were assessed for behavior pattern using the JAS and the SI. CHD patients were reported to show greater DBP elevations, compared to controls, independent of behavior type. SI-determined Type As demonstrated greater increases in SBP and DBP during the experimental task. Subjects assessed by the JAS failed to show significant differences on the physiological measures. This latter finding appears perplexing at first glance because both the SI and the JAS are related to CHD risk. Several explanations are possible. Some researchers would suggest that the JAS fails to as sensitive as the SI in detecting physiological differences (Matthews, 1982; Corse et al., 1985). It is also likely that the stressor was not of sufficient severity to induce greater reactivity in the JAS Type A subjects. Corse et al. (1982) suggested that for laboratory-induced stressors, the use of the SI may be more appropriate. Perhaps the use of extreme scores (ie., less than or greater than one standard deviation from the mean) for purposes of classification; may have resulted in different findings. Only future research will help to clarify these issues. The work of Williams & Lane (1982) may shed some light on this. These authors used both the SI and the JAS to

classify subjects (male college students) but only those classified by both techniques were used in the final sample. Two different tasks—mental arithmetic and reaction time—were used. Results showed that, during the mental arithmetic task, Type As showed greater muscle vasodilation, and enhanced secretion of epinephrine, norepinephrine, and cortisol than did Type Bs. The reaction time task was found to be associated with higher levels of testosterone in Type A subjects. This well designed study suggests that physiological differences can be found when using the JAS. It also points to the importance of task selection.

Results contradictory to the reactivity hypothesis were reported by Steptoe & Ross (1981). Using a series of cognitive tasks and the JAS student form, they assessed normal volunteers, and failed to find significant differences on cardiovascular measures (i.e., interbeat interval, pulse transit time, respiration, and galvanic skin response). In fact, Type Bs tended to show greater reactivity on some measures. This study also raises important questions about the appropriateness of the student form of the JAS. One should note also that the physiologic measures chosen have not been demonstrated to significantly differ in Type As and Bs in other studies.

A few studies using naturalistic settings or real-life stressors have been published. Endocrine reactivity (assessed via urinary epinephrine, norepinephrine, 17-ketosteroids, 17-hydroxycorticosteroids, and 5-hydroxyindole) was studied by Friedman, St. George, Byers, and Roserman (1960). SI-assessed

non-CHD male subjects collected urine during four consecutive workdays and upon awakening for four days. Results indicated that, at rest, catecholamine excretion did not differ. During the workday, only norepinephrine was found to be significantly related to Type A behavior. This study was probably among the first to demonstrate the lack of difference between As and Bs at rest, a finding which has been supported by later work. The lack of experimental control over the work situation and failure to measure workday stress are two important flaws in this study, and which are, perhaps, responsible for the lack of significance found on other variables.

The role of ACTH and cortisol was examined by Friedman, Roserman, and St. George (1969) and Friedman, Byers, and Roserman (1972). Following the injection of 100 units of ACTH (reported to be twice the amount needed to effect a maximal cortisol discharge), three urine samples were obtained (one immediately prior to injection, and one at 14 and 19 hours post-injection) so that analysis of 17-hydroxycorticosteroid (17-OHCS, a metabolite of cortisol and other adrenocortical glucocorticoid hormones) could be conducted. Subjects were 18 Type A and 20 Type B assessed by the early version of the SI. Results revealed no significant differences between basal levels of 17-OHCS. At 19 hours, considerable differences were found. Type As were found to have excreted abnormally low levels of 17-OHCS as compared to Bs. The hypo-respose of the Type As was perplexing. Friedman et al. (1969) postulated that this loss of "adrenal reserve" could be due

to a previous long standing or excessive discharge of ACTH. The authors suggested that future research, comparing ACTH levels in Type A and B subjects was necessary before conclusions could be drawn. As a follow up study, Friedman et al. (1972) measured plasma ACTH, plasma cortisol, and plasma cholesterol concentrations during five intervals of the working day in 9 Type A and 10 Type B subjects. Within-group and between-group cortisol and ACTH varied considerably and resulted in failure to find significant differences. Despite this, however, the authors noted that average ACTH values of Type A subjects was greater than those of Type B subjects at each of the six sampling intervals. Friedman et al. (1972) determined that there was a very low probability of observing these differences across all sampling intervals. They concluded from this that Type A subjects exhibited an increase in ACTH as compared to Type B subjects. Significance was found for the occurrence of greater peak values of ACTH in Type As. No significant differences were found for cortisol. As expected from their previous research, plasma cholesterol was significantly greater in Type As. Friedman et al. (1972) concluded that this hypersecretion of ACTH suggested that there were alterations in the hypothalamo-pituitary-adrenal systems of these Type A subjects. Despite obviously problematic methodology (i.e., small sample size, lack of experimental control), significant differences were found for ACTH, attesting, perhaps, to the robustness of this measure. However, the weaknesses in the study suggest that the results should be

interpreted with caution. Further research is necessary before definitive statements can be made. It is interesting to note that such research remains to be conducted.

A study conducted by Czeisler (1976) may help to shed some light on the difficulties inherent in utilizing endocrine responses as measures of reactivity. Four coronary patients awaiting open heart surgery and five healthy volunteers (hospitalized for purposes of the study) served as subjects. Czeisler (1976) collected plasma cortisol samples every 20 minutes for the 24 hours prior to the experimental groups' scheduled surgery. Anxiety was rated by each subject on a five point scale during each of these intervals. Indistinguishable and normal patterns of cortisol secretion were found for both the control and experimental groups across all sampling intervals with only one exception. During preoperative preparation for surgery (i.e., shaving, antiseptic wash, and enema), experimental subjects had 3.7 times higher cortisol concentrations than did controls ($p < .001$). In fact, cortisol in experimental subjects increased seven to 10 standard deviations from the control group mean obtained at the same time. No significant correlations were obtained between anxiety ratings and cortisol concentrations. These authors concluded that the experience of the pre-surgery situation alone did not result in hyperactivation of the pituitary-adrenal axis. Based on the results of cortisol secretion during preoperative preparation, Czeisler (1976) noted that, although cortisol is generated endogenously, it can be influenced by stress. Further,

frequent blood sampling was recommended by these authors because failing to do so in this study would probably have resulted in overlooking the significant data. Although the study reported by Czeisler (1976) did not address Type A reactivity, it does illustrate the level of complexity involved in psychoendocrine research.

A few other naturalistic studies are available in the current literature (Kahn, Kornfeld, Frank, Heller, & Hoar, 1980; Krantz, Arabian, Davia, & Parker, 1982; Kornfeld, Kahn, Frank, Heller, Freeman, & Keller-Epstein, 1985). All three of these studies address hemodynamic reactivity in patients undergoing coronary artery bypass grafting (CABG). Kahn et al. (1980) examined intraoperative blood pressure in 59 cardiac patients, assessed for behavior type using the SI. Blood pressure values were obtained by subtracting admission blood pressure (BP) from maximum intraoperative BP values. Results revealed significant correlations between SBP rise and the SI components of Overall Type A, Aggressive Content, and Job Commitment. Diastolic blood pressure and Job Commitment were also found to be correlated. These associations remained significant even when physical predictors of blood pressure rise were controlled. It is notable that significant hyperreactivity among Type A subjects was demonstrated despite patients being under general anesthesia. These findings were supported by Krantz et al. (1982). Using both SI- and JAS-determined Type A and B cardiac patients undergoing CABG, Krantz et al. (1982) found that, after controlling for

physical predictors of blood pressure rise, Type A (SI assessed only) was significantly related to SBP increases during surgery. Correlations with the JAS scales were smaller and unreliable. In addition, these authors reported a larger incidence of surgical complications (i.e., arrhythmias) in SI-assessed Type As. The JAS also failed to correlate with surgical complications. One should note, however, that the JAS analysis was conducted with an even smaller sample size ($N = 21$) than was originally used ($N = 27$). The results of Krantz et al. (1982) are consistent with previous research suggesting that conscious mediation is not necessary for Type A reactivity to occur. Disconfirming evidence was reported by Kornfeld et al. (1985), although non-cardiac patients undergoing general elective surgery served as subjects. Kornfeld et al. (1985) failed to find significant associations between SI-assessed Type A and intraoperative hemodynamic changes. A trend was noted for Overall Type A score and DBP at baseline and intraoperatively.

The importance of controlling for medications when studying cardiac patients has been demonstrated by several studies. Anxiety, at least in part, results from beta-adrenergic stimulation, and, therefore, drugs which reduce or block this response (i.e., beta-antagonistic drugs such as propranolol) would be expected to influence the hemodynamic and endocrine response to a stressful situation, particularly in subjects with somatic manifestations of anxiety. Beta-blockers are said to exert their greatest influence when sympathetic nervous system activity is

intense (Durel et al., 1985) suggesting the need for control when examining the stress response in cardiac patients. The early work of Granville-Grossman and Turner (1966) provided the first evidence that the use of propranolol was associated with a decrease in investigator-rated and personal symptom ratings of anxiety. Since this time, a number of studies have been conducted with beta-blocker treated cardiac patients under stress. A variety of different stressors have been used and results have not been conclusive. Some common results can be found across studies. Using normals and hypertensives exposed to a reaction time task, Heidbreder, Rockel, & Heidland (1978) found no effects of beta-blockade for HR. Different results were obtained by Bonelli, Hortnagl, Magometschnigg, Lochs, & Kaik (1979). These authors found a significant association between HR decrease and beta-blockade in normal volunteers' response to calculation stress. No effects were found for BP, E, or NE, although a very small sample size ($N = 6$) was used. Neftel, Adler, Kappeli, Rossi, Dolder, Kaiser, Bruggesser, & Vorkauf (1982) supported the findings from previous research that HR and beta-blockade were significantly related. However, again, no effect was found for catecholamines. Bonelli (1978) also found significantly reduced HR, as well as cardiac output, SBP and DBP reduced by beta-blockade. Bonelli (1978) also failed to find that beta-blockade significantly altered catecholamine response. One study that specifically addressed the association between Type A and beta-blockade was conducted by Krantz, Durel, Davia, Schaffer,

Arabian, Dembroski, & Macdougall (1982). Using a stressful interview and a history quiz, these authors found that propranolol treated patients showed less SI-assessed Type A characteristics, lesser HR, and lesser rate-pressure products, a correlate of myocardial oxygen supply. These effects were not found in patients being treated with nitrates, central nervous system drugs, or diuretics. No significant differences were found for BP. The JAS-assessed Type A behavior was not related to medication.

Although definite conclusions cannot be drawn from these studies, the evidence, in general, appears to support the notion that beta-blockade is useful in reducing some physiologic manifestations of anxiety (Durel et al., 1985). The most consistent effects appear to be on HR. There is still considerable controversy regarding BP and catecholamine response to stress and beta-blockade. A particular problem area appears to be the use of small sample sizes.

Several investigators have also examined the effects of calcium antagonists on physiologic reactivity (Corea, Miele, Bentivoglio, Boschetti, Agabiti-Rodei, & Muisesan, 1979; Taylor, Silke, Ahuja, & Ikoli, 1982; Heidbreder, Schafferhans, Kirsten, & Heidland, 1983; Pederson & Mikkelsen, 1978). Pederson and Mikkelsen (1978) reported significant decreases in HR and vascular resistance, and a significant increase in forearm blood flow, with acute administration of nifedipine in hypertensive patients. Corea et al. (1979) found a sustained decrease in BP (without HR

increase) in hypertensives. In normals, a significant increase in HR was found without any change in BP. In response to three different pressor stimuli, Taylor et al. (1982) documented a significant dose-related reduction in BP in six male hypertensives. Heidbreder et al. (1983) examined the physiologic response (via BP, HR, and catecholamines) to a mental stressor in normal persons taking either a calcium antagonist, nifedipine, or a diuretic, hydrochlorothiazide. The purpose of this study was to determine whether drugs with a hypotensive effect, but without sympatholytic properties, would result in the suppression of an emotional response to a stressor. The results of the study did not support this hypothesis and the authors concluded that neither drug inhibited emotional stress in normotensive subjects. The results of these studies are also controversial as are those investigating the effect of beta-blockade. Future reactivity research, controlling for the effect of medications, may shed some light on this confusion.

As can be seen from the foregoing review, findings within the area of Type and reactivity are not unequivocal. Although the greater majority of studies do show that Type As appear to be more reactive to stress than Type Bs, it is still a matter of speculation whether reactivity confers risk. It may also be obvious to the reader that several variables appear to influence reactivity. To summarize, characteristics of baseline, task (stressor), subject, and response measure assume critical importance. According to Houston and Ewart (1984), tasks that

provide a challenge, competition, or threat, have most often resulted in reactivity in Type A subjects. The use of such a wide variety of these parameters has resulted in some degree of confusion in the Type A literature. Future research aimed at isolating the important dimensions of these parameters is needed.

Summary

The Type A Behavior pattern, originally described by cardiologists Friedman & Rosenman (1959), is best characterized as a set of overt behaviors which can be elicited from a susceptible individual given an appropriately challenging environment (Matthews, 1982). The central elements of the behavior pattern appear to be a sense of time urgency, easily aroused hostility, and competitive achievement striving. Epidemiological studies, notably those of Rosenman et al. (1964, 1975) and Haynes et al. (1978, 1980), have shown that the Type A Behavior Pattern is significantly associated with an increased incidence of coronary heart disease, even when traditional risk factors are controlled. In 1981, the Review Panel on Coronary-Prone Behavior and Coronary Heart Disease concluded that Type A behavior is an independent risk factor for clinically apparent coronary heart disease and that the magnitude of risk associated with this behavior pattern is as great as those imposed by traditional risk factors such as elevated systolic blood pressure, smoking, and serum cholesterol.

As evidence began to accumulate in the area of Type A research, some authors began to speculate that psychophysiologic responsiveness (i.e., reactivity) could be a marker in the

development of cardiovascular disease (see review by Krantz et al., 1984). It was hypothesized that pathologic neuroendocrine and cardiovascular responses could link psychosocial stress to cardiovascular disease. A great deal of attention was then focused on the roles of the sympathetic adrenal-medullary system and the pituitary adreno-cortical system.

A variety of stressors have been used in the study of reactivity, including both laboratory (e.g., mental arithmetic, reaction time, cold pressor) and naturalistic (e.g., cardiac catheterizations, treadmill tests, and coronary bypass surgeries) settings. There is a substantial body of data to show that Type As and Bs fail to differ under conditions of rest. Under stress, however, Type As almost consistently show differences, as compared to Type Bs, on at least one of the neuroendocrine and/or cardiovascular measures under study. Under conditions of stress, Type As, as compared to Type Bs, tend to show larger episodic increases in blood pressure, heart rate, catecholamines, ACTH, and cortisol. It is noteworthy that larger differences are often found when real-life stressors are used, attesting to the importance of task selection in this area of study.

Several shortcomings within the area of reactivity were noted in the foregoing review. First, although a great deal of research has assessed the catecholamine (e.g., norepinephrine, epinephrine) response to stress, little attention has been paid to the release of adrenocorticotrophic hormone (ACTH) in response to stress. Only two studies, published in the late 1960s and early 1970s, were

found in the literature. In comparison to studies that examined catecholamines and cortisol, these two ACTH studies reflect a deficit in the area. Further, recall that pituitary ACTH cannot be assumed from measures of cortisol concentration (Krieger, 1979). It would appear then that little is known about ACTH reactivity in Type A subjects. This represents a major gap in the literature, particularly in light of the importance of ACTH in the stress response. ACTH is a well documented indicator of stress (Axelrod, 1984) and the present study utilizes this measure as a physiological dependent variable. Second, the use of laboratory stressors dominates the area and several researchers have suggested the need for more naturalistic studies (Krantz et al., 1984). Further, the JAS has been infrequently used, or has been used with small samples, such that less is known about JAS-assessed Type A responses to a challenge or stressor. The objectivity of the JAS, as well as some practical considerations, suggests that its use in Type A assessment will continue so that the importance of establishing its parameters is necessary. The present study compares the responses of JAS-assessed Type As and Bs to a real-life cardiac catheterization. Finally, until recently there has not been adequate attention placed on the importance of medications and how they might alter the stress response. Existing research has thus far been inconclusive. Because of a number of practical problems, the most important of which was sample sizes, it was not possible in the present study to form different drugs groups. The next best approach, that of

comparing patients on target medications (i.e., beta-blockers, calcium-channel blockers or both of these) to those that were not on target medications, was used. In addition to the advantage of controlling these variables, the present study also attempted to investigate the effects of these medications on the response to stress.

Purpose of the Present Study

The purpose of the present study was to investigate how cardiac patients, identified as manifesting Type A or Type B Behavior Patterns, would respond to the stress of cardiac catheterization. The primary research question being asked was whether the Type A cardiac patients would show an increased physiological and psychological reactivity (i.e., exaggerated response) to the stressor. Limited ACTH research thus far would suggest that Type A individuals are indeed more reactive to stress than their counterparts, the Type B individual. However, published findings (Friedman et al., 1969, 1972) have been few and not without controversy. One of the important aims of the present study, therefore, was to aid in the clarification of this phenomenon, particularly with respect to the role of ACTH in the response to stress. Another important research question addressed in the present study was that of how medications might influence or alter the response to a stressor. Patients in the study were divided into two medication groups. Patients taking beta adrenergic blockers, calcium entry blockers, or both of these drugs formed the drug condition of the independent variable of

Drug Group. Patients not taking any of these medications formed the no drug condition of Drug Group.

Based on the findings from previous research, several hypotheses were formulated.

1. All subjects are expected to show some degree of hormonal (i.e., ACTH) and cardiovascular (i.e., HR and BP) responsivity to the stress of cardiac catheterization. Although individual differences will exist, there is substantial data to suggest that exposure to stress results in the mobilization of the sympathetic-adrenal-medullary system and the pituitary-adrenal-cortical system (Axelrod et al., 1984). This is said to be particularly true when a stressor has an element of uncertainty or unpredictability (Mason, 1968) or when it results in fear and anxiety (Selye, 1950). The cardiac catheterization can be said to possess many of these elements, and therefore, aside from individual differences that might exist, a general response to stress can be expected across all subjects.

2. Numerous studies previously reviewed (e.g., Krantz et al., 1982; Eliot, 1982) suggest that Type A individuals, in comparison to Type Bs, tend to show exaggerated responsivity (i.e., reactivity) to stressful situations. Although some disconfirming evidence has been reported, Type A subjects have been shown to respond to stress with greater elevations in HR and BP (Dembroski et al., 1977; VanEgeren, 1979; Hart et al., 1983; Corse et al., 1982; and Kahn et al., 1980) and ACTH (Friedman et al., 1969, 1972). Therefore, Type A subjects in the present study

are expected to show greater hormonal and cardiovascular reactivity than Type B subjects.

3. Drug Group (i.e., drug versus no drug) is also expected to influence reactivity to stress. Although again research in this area has not been without controversy, the findings reported by Bonelli (1978, 1979) and Krantz et al. (1982) suggest that beta-blockade (i.e., treatment with beta-adrenergic blocking drugs) is useful in reducing physiologic manifestations of anxiety. Durel et al. (1985) note that the most consistent finding is that of HR reduction. With respect to calcium-channel blockers, evidence is even more sparse and equally conflictual. Several researchers (Pederson et al., 1978; Corea et al., 1982; Taylor et al., 1982) have reported that reductions in HR and BP occur in patients treated with calcium-channel blockers. Conversely, Heidbreder et al. (1983) reported that treatment with these drugs does not suppress the emotional response to a stressor. No data appear to be available with regard to the combined treatment of both beta-blockers and calcium-channel blockers. However, clinical observation (Usher, 1985) suggests that the effect of the beta-blocker will "override" the effect of the calcium-channel blocker. Therefore, the predicted primary effect of the Drug Group is that of beta-blockade and consequential stress reduction. Subjects in the No Drug condition are expected to show the greatest response to the stressor. The hypothesis related to the influence of drug group upon Behavior Type is illustrated as follows: Type A (neither drug) > Type A

(drug) > Type B (neither drug) > Type B (drug).

4. Regarding psychological measures (i.e., anxiety and mood scales), similar events are hypothesized. Following the logic proposed in the preceding hypotheses, all subjects are expected to show some degree of psychologic responsiveness. Type A subjects, as compared to Type B subjects, are expected to demonstrate exaggerated responsiveness and medications are expected to influence this response. That is, patients in the Drug Group are expected to report less anxiety and mood disturbance because of the perception of less somatic manifestation of anxiety produced by these drugs. Further, subjects showing elevated trait anxiety scores are predicted to demonstrate greater psychological and physiological responsiveness.

CHAPTER TWO

Method

Subjects

Sixty-three patients of the Medical University Hospital in Charleston, SC, referred for cardiac catheterization from November-June, 1986, served as subjects in the present study. Partial data was also collected on an additional 21 subjects that could not be included in data analysis. Eleven of these 21 subjects were not available for post-catheterization follow-up because of other scheduled diagnostic tests or because of the necessity for emergency surgery. Approximately six subjects were lost because of experimenter error. An additional two subjects were excluded because of conditions unknown to the experimenter at the time of data collection (i.e., pacemaker and dialysis patient). One subject was excluded because of an invalid JAS (i.e., greater than six items unanswered). Another subject did not undergo scheduled catheterization. Criteria for inclusion in the study were: (a) English-speaking white males between the ages of 25 and 70 referred for diagnostic workup for symptoms of coronary heart disease (eg., angina, myocardial infarction); (b) consent of primary physician; and (c) willingness to participate in the study. Patients were excluded from the study if they had (a) previous coronary surgery; (b) diseases known to affect ACTH production (e.g., Cushing's disease); (c) cardiovascular physiology was controlled by artificial means (e.g., pacemaker), or (d) if they were taking long term psychotropic medications.

Independent Variables

The Type A Behavior Pattern was assessed using the Jenkins Activity Survey-Form C (JAS, Jenkins et al., 1979). The JAS was computer scored and standard and percentile scores were obtained to yield an Overall Type A score and three factor scores— Speed and Impatience, Job Involvement, and Hard-Driving and Competitive. Subjects were designated as Type A or Type B according to a median split using the Overall Type A standard score. That is, subjects scoring in the positive direction (i.e., greater than the mean of 0.00) were identified as Type A subjects. Type B subjects were those with Overall Type A scores in the negative direction (i.e., less than the mean of 0.00).

Subjects were also be grouped according to what type of medication they were taking for their coronary symptoms. Within each of the two behavior groups (A or B), two medication groups were formed: (a) patients taking beta-adrenergic blocking agents (e.g., propranolol), calcium entry blocking agents (e.g., nifedipine, and patients taking both of these drugs; and (b) patients taking neither of these two drugs.

Dependent Variables: Physiological

All dependent measures were obtained on each of two occasions for each subject—approximately one hour prior to catheterization (stress condition) and the morning after the catheterization (non-stress condition), 24 hours after pre-catheterization measures were obtained.

The major dependent variable assessed in the study was level

of plasma adrenocorticotrophic hormone (ACTH). Two fasting blood samples were obtained as described above. ACTH samples were analyzed using Nichols Institute Diagnostics commercial ACTH radioimmunoassay (RIA) kits. For the present experiment, 10.0 milliliters of venous blood was collected in siliconized glass tubes, with EDTA added as a anticoagulant, on each of two occasions (i.e., pre- and post-catheterization). To prevent enzyme degradation, blood samples were kept on ice until centrifuged and frozen. Plasma was separated after samples had been centrifuged at 2200 x gravity, 4 degrees centigrade for 15 minutes and at 7500 x gravity, 4 degrees centigrade, for 10 minutes. Plasma samples were stored at -80 degrees centigrade until assay.

The radioimmunoassay procedure is based upon the principles of competitive binding established by Berson et al. (1968). The basic principle involves the measurement of unlabeled ACTH (i.e., unknown sample) by its ability to compete with radiolabeled ACTH for specific antibody binding sites. Known concentrations of ACTH (i.e., standard samples provided in the kit) are used to establish a dose-response curve. Patient samples are then evaluated by comparison with this curve. As concentrations of unlabeled ACTH increase, the amount of radiolabeled ACTH bound to antibody decreases proportionately. In a sample of 278 healthy adults, Nichols Institute (kit manufacturer) reported the normal range of ACTH to be less than 130 pg/ml. Normal values are also routinely established by each laboratory as one of the many accuracy checks

involved in the RIA. Using 13 healthy adults, the present investigator found that all values were below 130 pg/ml (\bar{M} = 42 pg/ml, SD = 37.00).

The instructions for the radioimmunoassay were followed exactly with the exception of an addition of two other standard samples. All samples were assayed in duplicate and pre and post samples for each patient were analyzed in the same assay. All samples were counted in a Model 1282 Gammacounter (Wallace Instruments, Inc.). Samples with counts per minute exceeding that of the maximum binding tube were incalculable and were excluded from the analysis (n = 12). Therefore, only 48 ACTH samples on each Day 1 and Day 2 are included in ACTH analyses. It is not known what produced the enhanced binding in these subjects.

Reliability of plasma ACTH radioimmunoassays has been reported by Berson et al. (1968). When duplicate samples are assayed during the same procedure, levels of agreement have been reported to be excellent. Somewhat less agreement has been observed when duplicate samples are assayed in two different procedures. In the present study, duplicate samples were analyzed in the same assay.

In order to assess group differences in level of arousal, other physiologic measures reflecting anticipatory anxiety were obtained. These were blood pressure (as measured by sphygmomanometry) and heart rate (as measured using radial pulse) obtained once under both conditions of stress and non-stress as described above. For each subject, each of these measurements was

taken using the same arm and with the patient in the same physical position (i.e., lying in bed).

Dependent Variables: Psychological

Two psychological measures were used to assess subjects level of perceived stress. The State-Trait Anxiety Inventory-Form X (STAI, Spielberger, Gorsuch, & Levine, 1970) and the Profile of Mood States (POMS, McNair, Lorr, & Droppleman, 1971) were administered to all subjects under both conditions of stress and non-stress. The State portion of the STAI and the POMS were used to assess current level of arousal. The Trait portion of the STAI was used in order to investigate associations between self-reported trait anxiety and measures of reactivity.

The STAI is composed of two separate self-report scales for measuring state anxiety (A-State) and trait anxiety (A-Trait). The A-State portion of the inventory contains 20 statements to which the subject is instructed to respond according to how he or she feels at that particular moment. The A-Trait portion is similar, however, the subject is instructed to respond according to how he or she generally feels. A four-point scale (not at all, somewhat, moderately so, and very much so) is used on both inventories. Test-retest reliability for the A-Trait has been found to range from .73 to .86. A-State test-retest reliability is much lower (i.e., .16 to .54) as would be expected since moment to moment anxiety levels are likely to fluctuate. Internal consistency has also been demonstrated with reliability coefficients ranging from .83 to .92. Construct and concurrent

validity have also been demonstrated (see Spielberger et al., 1970).

The POMS is a 65-item adjective rating scale in which the subject is instructed to use a five-point scale to indicate how he or she has been feeling during the past (time set). For purposes of the present study, a time set of Right Now was used. The five-point rating scale is composed of: not at all, a little, moderately, quite a bit, and extremely. Six factor scores are derived: (a) Tension-Anxiety, (b) Depression-Dejection, (c) Anger-Hostility, (d) Vigor-Activity, (e) Fatigue-Inertia, and (f) Confusion-Bewilderment. A Total Mood Disturbance (TMD) score was computed by summing all scores except Vigor-Activity. This latter factor score was subtracted from the sum of the other factor scores resulting in the TMD score. Only the TMD score was used in the present analyses. For a more thorough description of these factor scores, the reader is referred to McNair et al. (1971). Both the rest-retest reliability (ranging from .61 to .69) and internal consistency (.90 and above) estimates have been highly satisfactory. Evidence for the predictive and construct validity of this self-report instrument has been demonstrated in brief psychotherapy studies and controlled outpatient drug trials.

Procedure

Patients scheduled for cardiac catheterization are usually hospitalized for 36-48 hours. Prior to hospital admission, all patients were provided with an information booklet (Purcell, 1982) which described the catheterization procedure in simple terms.

Following admission, potential subjects were recruited by the principal investigator. Patients were told that the present study was designed to investigate how different individuals responded to the stress of cardiac catheterization. After signing an approved informed consent agreement (see Appendix A), subjects completed a Personal Information Form (see Appendix B) which requested demographic information (age, education, occupation) and information regarding traditional risk factors (e.g., family history of cardiovascular disease, smoking history, etc.). The JAS and the Trait portion of the STAI were also completed at this time. Subjects were also seen on this day by the attending cardiologist or the Cardiology Fellow in order to answer any remaining questions that the subject (patient) may have had regarding the cardiac catheterization.

On the morning of the procedure, prior to sedation with an oral dose of secobarbital 100mg, vital signs (BP and HR) and blood samples were obtained by a registered nurse on the unit. The STAI and the POMS were completed by the subjects at this time. These measures were obtained, as closely as possible, at approximately one hour prior to scheduled time of catheterization. Delays and/or changes in expected time of catheterization did occur due to procedures followed by the catheterization team. However, once measures were collected, these subjects were included in the data analysis unless they became unavailable for post-catheterization data collection procedures (e.g., next day emergency surgery). No attempt was made to assess fear of the blood-drawing procedure.

However, a number of patients refused to participate in the present study stating this fear as the primary reason for noncompliance with the study.

All catheterizations were performed by the Fellow in Cardiology under the supervision of the attending cardiologist. Standard clinical cardiac catheterizations were done. Catheterizations for subjects in the present study were limited to those scheduled between the hours of 7:00 a.m. and 12:00 p.m. Catheterizations were conducted Monday through Friday. All patients were again seen by their physician on the evening of the catheterization for a report of preliminary findings and recommendations. It was impossible to alter this routine without disrupting the standard procedure followed by the catheterization team. Therefore, this information was taken into account in the statistical analyses.

On the day after the catheterization, 24 hours after dependent measures were obtained the previous day, fasting blood samples and vital signs were obtained and the STAI and the POMS were again completed.

Each subject's medical chart was also reviewed in order to obtain information on traditional cardiovascular risk factors, medications, and catheterization results and recommendations. Catheterization recommendations were recorded as (a) surgery necessary, (b) medication only, and (c) other. The category of other was used to indicate a more general recommendation of further procedures (i.e., coronary angioplasty or a trial of

medication followed by surgery if unsuccessful).

Upon completion of data collection, subjects were debriefed and thanked for their participation.

Experimental Design and Statistical Analysis

A 2 x 2 x 2 (Behavior Pattern x Drug Group x Day) mixed effects design was employed. Two levels of behavior pattern (Type A or Type B) and two levels of drug group (drug and no drug) were used. Day served as the repeated factor. There were two levels of day: pre-catheterization (Day 1) and post-catheterization (Day 2). Day 2 was conceptualized as the best available assessment of baseline functioning. Initially, the study was designed to investigate four drug groups: beta-blockers, calcium channel blockers, a combination of both of these, and a group of subjects who did not take either of these two medications. Due to small sample sizes, in general, and the loss of 12 ACTH samples, in particular, it was deemed that the use of four drug groups would be impractical. Therefore., drug groups were collapsed to form two levels of the independent variable of drug group. The drug condition (level) was composed of patients taking beta-blockers, calcium-channel blockers, or a combination of these. The no drug condition was comprised of patients taking neither of these two drugs.

CHAPTER THREE

Results

A total of 63 subjects were available for analysis. After means and standard deviations were calculated, an analysis of outliers was conducted. A total of 15 subjects were found to fall at least two standard deviations from the mean on at least one dependent variable. Removal of 15 subjects was deemed to be impractical as it would have greatly reduced sample size to unacceptable levels. Therefore, only those subjects with scores of at least three standard deviations from a mean of a particular dependent variable mean were excluded. Outliers on any one dependent variable were excluded from all analyses. After three subjects were excluded, the final sample was 60. Statistical analyses were conducted on the full sample ($N = 60$) and on a subsample of this population referred to as the extreme sample ($N = 30$). The extreme sample was composed of subjects from the full sample who scored in the upper (i.e., extreme Type A) and lower (i.e., extreme Type B) quartiles on the Overall Type A scale of the JAS.

Full Sample

Subjects ranged in age from 37 to 69 years ($M = 53.45$, $SD = 9.68$, $N = 60$). Additional demographic data is presented in Table 1. Twenty-eight Type As and 32 Type Bs were identified. Means and standard deviations for the four JAS scales are presented in Table 2.

Chi-square analysis revealed no significant differences

Table 1

Demographic Characteristics of Sample (N= 60)

<u>Variable</u>	<u>Per Cent</u>
Education	
< 12 years	23.0
high school	25.0
some college	20.0
college degree	32.0
Employment	
full-time	56.0
part-time	3.0
unemployed	2.0
retired	38.0
History of Myocardial Infarct	48.0
Family History of CHD	58.0
Hypertension	35.0
Diabetes	15.0
Hyperlipidemia	10.0

Table 2

Mean Standard Scores of Jenkins Activity Survey (JAS) Factors for
Type A (n=28) and Type B (n=32) Subjects

<u>JAS</u>	<u>Type A</u>	<u>Type B</u>
Overall Type A	<u>M</u> = 9.30	<u>M</u> = -8.37
	<u>SD</u> = 6.25	<u>SD</u> = 3.96
Speed & Impatience	<u>M</u> = 6.16	<u>M</u> = 6.82
	<u>SD</u> = 6.57	<u>SD</u> = 8.85
Job Involvement	<u>M</u> = -1.24	<u>M</u> = -9.85
	<u>SD</u> = 11.25	<u>SD</u> = 8.11
Hard-Driving & Competitive	<u>M</u> = 4.50	<u>M</u> = -0.91
	<u>SD</u> = 10.06	<u>SD</u> = 10.80

Note. The JAS factors are scored such that the normative means are 0.00 and the standard deviations are 10.00.

between behavior type or drug group for age, education, occupational status, history of myocardial infarction, family history of CHD, diabetes, hyperlipidemia, or cigarette smoking. Presence or absence of hypertension was found to be significant for drug group, $X^2(3, N = 60) = 9.06, p < .03$.

A one-way ANOVA was also conducted for catheterization recommendations (i.e., surgery, medication, other) to examine whether knowledge of recommendations influenced the dependent variables obtained on day 2. No significant differences were found.

A $2 \times 2 \times 2$ (Behavior Type \times Drug Group \times Day) analysis of variance (ANOVA) with repeated measures on day was performed for all dependent variables using both the full sample ($N = 60$) and the extreme sample ($N = 30$). Dependent variables were adrenocorticotrophic hormone (ACTH), systolic blood pressure (SBP), diastolic blood pressure (DBP), state anxiety, and total mood disturbance (TMD). Statistical summary tables for the full sample are presented in Appendix C. Statistical summary tables for the extreme sample are presented in Appendix D.

Using the full sample, a significant main effect for Day was revealed for ACTH, $F(1, 44) = 6.38, p < .01$. Mean ACTH concentrations for day 1 (pre-catheterization day) and day 2 (post-catheterization day) were 43.17 pg/ml (picograms per milliliter of plasma) and 34.57 pg/ml, respectively. A significant main effect for drug group was also revealed for ACTH, $F(1, 44) = 4.42, p < .04$. Subjects in the drug condition exhibited a mean

ACTH level of 35.33 as compared to a mean of 47.48 for subjects in the no drug condition. A significant Type x Drug interaction was found for ACTH, $F(1, 44) = 9.63, p < .003$. The interaction is illustrated graphically in Figure 1. Relevant means and standard deviations are presented in Table 3. Inspection of Figure 1 reveals that Type B subjects did not differ regardless of drug condition. In contrast, Type A subjects exhibited substantially larger ACTH concentrations in the no drug condition as compared to the drug condition. Further, Type A subjects on no drugs exhibited much larger responses to the stressor than did Type B subjects on no drugs.

In terms of the full sample, a main effect for Day was found for SBP, $F(1, 56) = 4.51, p < .04$. Systolic blood pressure on day 1 was 122.78 mmHg (millimeters of mercury) as compared to 118.68 mmHg for subjects on day 2. A Type x Day interaction was also revealed for SBP, $F(1, 56) = 6.39, p < .01$. The interaction is graphically illustrated in Figure 2. Means and standard deviations for this interaction are presented in Table 4. Inspection of Figure 2 reveals that SBP for Type As and Type Bs did not differ on day 1. In contrast, Type As demonstrated substantially lower levels of SBP, as compared to Type Bs, on day 2.

No significant main effects or interactions were revealed for DBP, HR, or TMD. The only effect revealed for state anxiety was a main effect for day, $F(1, 56) = 3.97, p < .05$. This main effect for day reflected a significant difference between 36.02 for day 1

Figure 1. Means of ACTH Collapsed across Days by Drug Group For Type A and Type B Subjects ($N = 48$)

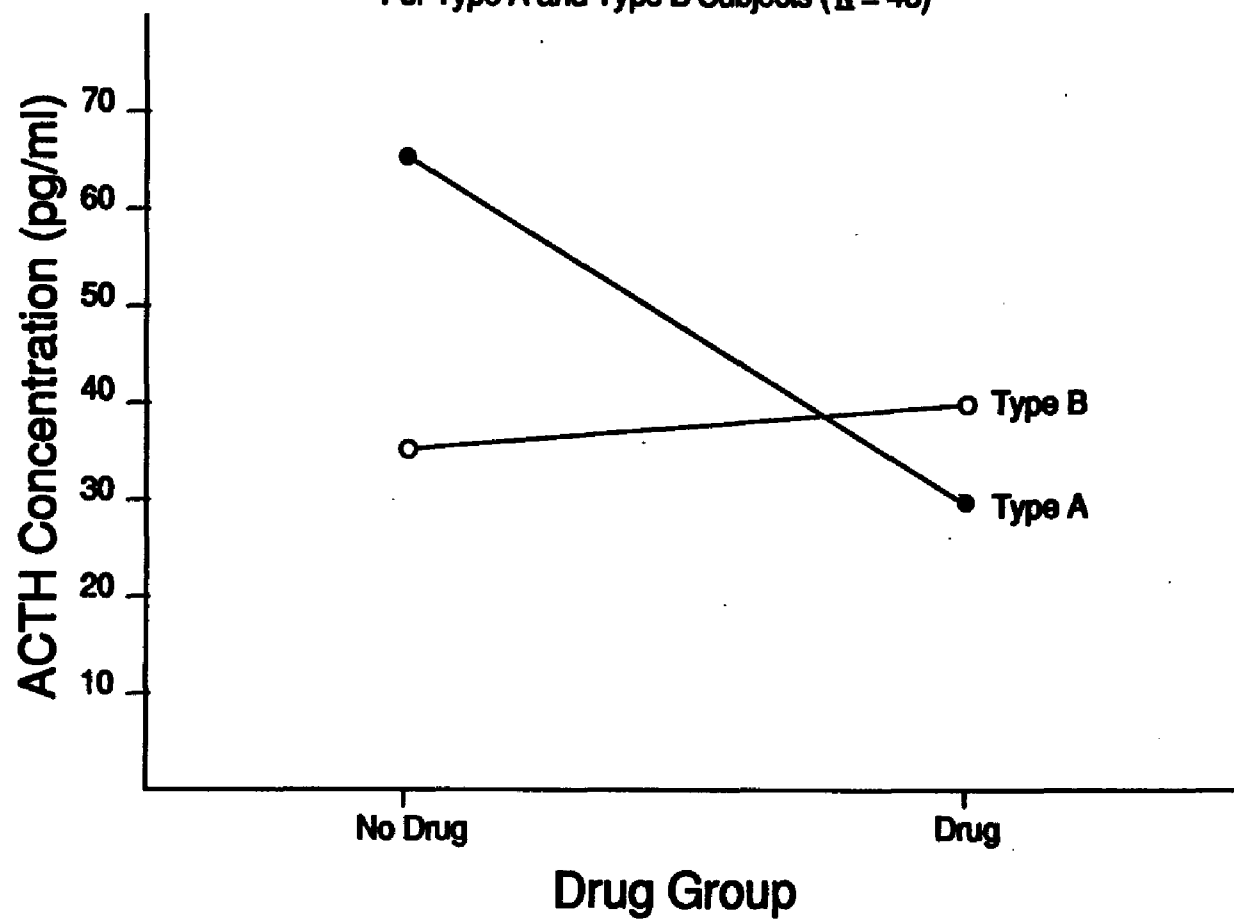


Table 3

ACTH Means and Standard Deviations for Type x Drug x Day

(N = 48)

<u>n</u>	<u>Type</u>	<u>Drug</u>	<u>Day</u>	<u>M</u>	<u>SD</u>
6	A	no drug	1	77.87	34.40
6	A	no drug	2	54.14	31.01
16	A	drug	1	34.33	21.41
16	A	drug	2	25.18	13.50
8	B	no drug	1	34.38	12.08
8	B	no drug	2	32.78	17.68
18	B	drug	1	43.37	33.10
18	B	drug	2	37.20	27.34

Figure 2. Means of SBP Collapsed across Drug Groups by Day For Type A and Type B Subjects ($N = 60$)

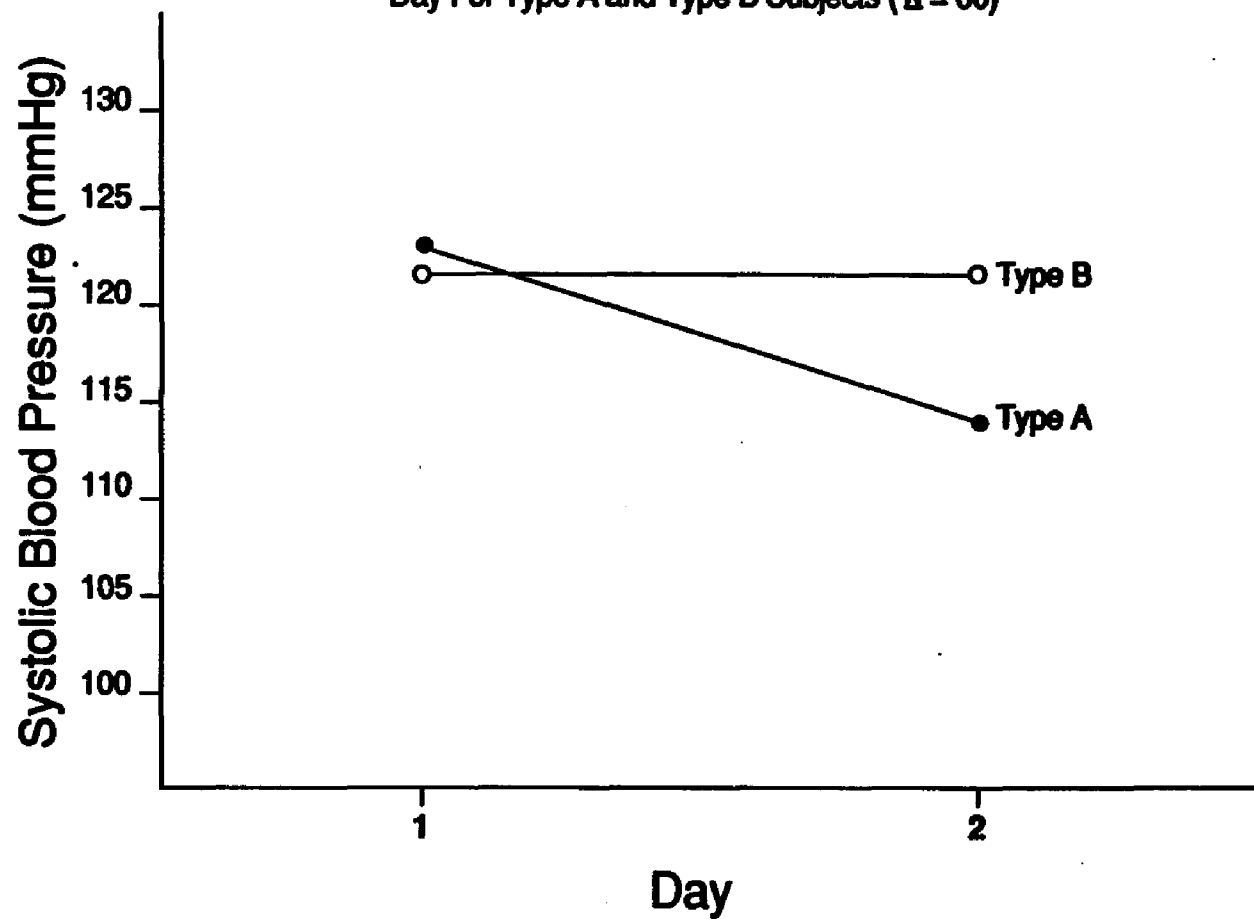


Table 4

SBP Means and Standard Deviations for Type x Drug x Day(N = 60)

<u>n</u>	<u>Type</u>	<u>Drug</u>	<u>Day</u>	<u>M</u>	<u>SD</u>
8	A	no drug	1	128.62	32.71
8	A	no drug	2	112.25	10.71
20	A	drug	1	121.25	16.94
20	A	drug	2	115.50	13.84
10	B	no drug	1	121.20	16.73
10	B	no drug	2	124.80	21.56
22	B	drug	1	122.77	15.50
22	B	drug	2	121.14	12.19

and 33.93 for day 2. Means and standard deviations for State Anxiety, DBP, HR, and TMD are presented in tables 5, 6, 7, and 8, respectively.

Extreme Sample

A 2 x 2 (Behavior Type x Day) ANOVA with repeated measures on day was conducted for the extreme sample ($N = 30$). No main effects for type or Type x Day interactions were revealed for any of the dependent variables. Two significant main effects for day were found for HR, $F(1, 28) = 4.30$, $p < .05$, and for state anxiety, $F(1, 28) = 4.07$, $p < .05$. The means for HR for this extreme sample were 68.40 and 71.07 for day 1 and day 2, respectively. For state anxiety, the main effect for day represents a significant difference of 36.23 for day 1 and 33.03 for day 2.

Correlational Data

Pearson correlations were obtained among all dependent variables. The correlation matrix is presented in Table 9. For purposes of clarity of presentation, dependent measures will be classified as (a) biochemical, including ACTH; (b) cardiovascular, including SBP, DBP, and HR; or (c) psychological or self-report, including State Anxiety and TMD. Inspection of the matrix reveals that the agreement between each dependent variable on day 1 and day 2 was highly significant. For example, ACTH on Day 1 was highly related to ACTH on Day 2 ($p < .001$). With only one modest exception, no significant associations were revealed between the three response classes. That is, ACTH (day 1

Table 5

DBP Means and Standard Deviations for Type x Drug x Day(N = 60)

<u>n</u>	<u>Type</u>	<u>Drug</u>	<u>Day</u>	<u>M</u>	<u>SD</u>
8	A	no drug	1	76.25	11.83
8	A	no drug	2	72.00	8.28
20	A	drug	1	76.20	10.93
20	A	drug	2	75.60	8.45
10	B	no drug	1	77.40	7.24
10	B	no drug	2	79.00	8.60
22	B	drug	1	76.27	10.15
22	B	drug	2	77.54	10.79

Table 6

HR Means and Standard Deviations for Type x Drug x Day(N = 60)

<u>n</u>	<u>Type</u>	<u>Drug</u>	<u>Day</u>	<u>M</u>	<u>SD</u>
8	A	no drug	1	68.50	7.54
8	A	no drug	2	65.75	9.10
20	A	drug	1	68.45	10.44
20	A	drug	2	72.45	11.01
10	B	no drug	1	72.60	6.99
10	B	no drug	2	76.70	8.87
22	B	drug	1	67.23	11.86
22	B	drug	2	69.32	7.85

Table 7

State Anxiety Means and Standard Deviations of Type x Drug xDay (N = 60)

<u>n</u>	<u>Type</u>	<u>Drug</u>	<u>Day</u>	<u>M</u>	<u>SD</u>
8	A	no drug	1	36.25	6.54
8	A	no drug	2	32.25	7.40
20	A	drug	1	36.80	10.89
20	A	drug	2	35.10	9.01
10	B	no drug	1	36.70	11.45
10	B	no drug	2	34.20	10.81
22	B	drug	1	34.90	9.31
22	B	drug	2	33.36	9.10

Table 8

TMD Means and Standard Deviations of Type x Drug x Day(N = 60)

<u>n</u>	<u>Type</u>	<u>Drug</u>	<u>Day</u>	<u>M</u>	<u>SD</u>
8	A	no drug	1	150.50	32.74
8	A	no drug	2	148.62	33.96
20	A	drug	1	149.15	28.01
20	A	drug	2	140.05	27.25
10	B	no drug	1	149.90	26.70
10	B	no drug	2	147.60	20.22
22	B	drug	1	150.27	36.98
22	B	drug	2	144.86	31.00

Table 9

Pearson Correlation Matrix for all Dependent Variables

	ACTH1	SBP1	DBP1	HR1	State1	TMD1	ACTH2	SBP2	DBP2	HR2	State2	TMD2
ACTH1												
SBP1	.08											
DBP1	-.08	.63***										
HR1	-.26	.11	.13									
State1	.07	.00	-.03	.23								
TMD1	.14	-.01	-.05	.22	.64***							
ACTH2	.58***	-.02	-.03	-.22	.11	.14						
SBP2	-.14	.49***	.36**	.27*	.17	.16	-.00					
DBP2	-.09	.39**	.50***	.27*	.16	.24	-.09	.71***				
HR2	-.32**	.15	.21	.61***	.28*	.21	-.20	.05	.19			
State2	.01	.12	-.03	.20	.59***	.50***	-.04	.27*	.16	.13		
TMD2	.04	.06	-.02	.28*	.55***	.70***	.07	.19	.18	.27*	.78***	

* $p < .05$, ** $p < .01$, *** $p < .001$ Note. All significance levels were obtained using two-tailed tests.

or day 2) did not correlate with any of the cardiovascular or psychological measures. Similarly, cardiovascular measures on day 1 were not related to psychological measures on day 1. Two significant, but modest, correlations were revealed between cardiovascular and psychological measures on day 2. That is, state anxiety (day 2) was related to SBP (day 2). TMD (day 2) was modestly associated with HR (day 2). Regarding associations among dependent variables within the same classification, it was found that (a) both self-report measures (state anxiety and TMD) correlated highly with one another on day 1 and day 2; and (b) measures of SBP and DBP on day 1 and day 2 were highly related, but no significant correlations were revealed for HR. With respect to trait anxiety, significant correlations were revealed with state anxiety on day 1 and day 2 ($p < .0001$) and with TMD on day 1 and day 2 ($p < .0001$). No significant correlations for trait anxiety were noted among the biochemical measures or the cardiovascular measures.

CHAPTER FOUR

Discussion

The present study was designed to investigate physiological and psychological reactivity to stress in JAS-assessed Type A and Type B cardiac patients. The evaluation of the effects of medications on the response to stress was also an integral goal of the current study. Briefly, and with regard to the full sample ($N = 60$), significant day main effects were revealed for ACTH, SBP, and state anxiety. A significant main effect for drug and a significant Type x Drug interaction was found for ACTH. A significant Type x Day interaction was revealed for SBP. With regard to the extreme sample ($N = 30$), significant main effects for day were found for state anxiety and HR. Because a different pattern of results were noted for the full sample and the extreme sample, these findings will be discussed separately.

Full Sample

It was hypothesized that all patients would perceive the catheterization day (day 1) to be more stressful than the post-catheterization day (day 2). The main effects for day, found for ACTH, SBP, and state anxiety, provide support for this hypothesis. Patients, as a group, did respond to day 1 with higher levels of ACTH, SBP, and anxiety ratings as compared to day 2. Although the mean differences between day 1 and day 2 on these three measures were relatively small, the sample size used in the present study suggests that these differences were important and noteworthy. The data indicate, then, that the cardiac

catheterization did serve as a significantly stressful event for the subjects.

It was predicted that Type As, as compared to Type Bs, would exhibit greater responsiveness to the catheterization procedure. This hypothesis was not supported in the present study. This failure to find differences between Type A and Type B subjects is inconsistent with much of past research (e.g., Kahn et al., 1980; Corse et al., 1982; and Krantz et al., 1982). Consistent with the present results are those studies from which negative findings have been published (e.g., Steptoe et al., 1981; Case et al., 1985). A number of possibilities exist for this failure to find A-B differences. First, there is some evidence to suggest that age may be an important factor in reactivity to stress (Houston & Ewart, 1984; Watkins & Eaker, 1986). The research in this area is controversial but most of the available data indicates that, with increasing age, there is decreasing sensitivity to beta-adrenergic stimulation. This data would imply that, for at least cardiovascular measures of reactivity, older individuals may appear less responsive to stress. The association between ACTH and age is more speculative. Because the size of the pituitary decreases with age, there may be a correlation between increasing age and decreasing ACTH secretion (Daughaday, 1981). The sample used in the current study was comprised largely of older individuals ($M = 53$ years). The use of an older population may have restricted the range of responses to the stressor. It is possible that a younger sample may have exhibited more pronounced A-B differences.

This would imply, as has been recommended recently in the literature, that age factors should be more closely examined in future reactivity research (Houston et al., 1984; Watkins et al., 1986).

A second possible source of influence in the current data set involves the effects that drugs (i.e., beta-blockers, calcium-channel blockers, and both of these) had on the response to the stressor. The use of these drugs were associated with lower ACTH responses in Type A individuals to levels consistent with those shown by Type B subjects. Because the greater proportion of the study subjects were taking these target drugs, it is possible that these drugs may also have restricted the range of possible stress responses and/or served to further mask the differences between Type As and Type Bs. It should also be noted that the effects of some drugs (e.g., nitrates) were not systematically controlled. It is possible that the effects of uncontrolled medications also affected the current findings. The finding that drugs were associated with significantly reduced responsiveness to the stressor strongly suggests that future reactivity research control for these effects.

The failure in the current study to use extreme behavior types may also have contributed to the lack of A-B differences. There has been some suggestion in the literature (Krantz et al., 1984) that extreme Type As are more reactive to stress than non-extreme Type A individuals. The majority of this research has been shown with the Structured Interview. The use of extreme

scores may be necessary to obtain more salient differences between Type A and Type B subjects. An attempt was made to explore this possibility in the current study by conducting an additional analysis of the dependent measures of those subjects scoring in the extreme Type A and B range. The failure to find significant A-B differences in this case may have resulted from the small sample that was used ($N = 30$).

One final area of concern with respect to reasons for failing to identify significant A-B differences involves those patients who refused to participate in the study. These patients, approximately 15-20 in number, refused to participate for reasons that, on an unsystematic observation, appeared anxiety-related (e.g., fear of needles, too much to deal with during hospitalization, too much paperwork, etc). The inability to include these patients, from whose verbal behavior one might speculate anxiety and/or time urgency, may have also restricted the range of responses to the stressor. That is, Type As, because of their impatient nature, may have been less willing to participate in a time-consuming study. This would hold especially true for extreme Type As that would be expected to harbor the most intense feelings of time urgency. Future research aimed at investigating individuals who refuse to participate in studies would be helpful in determining possible biases in study populations.

The speculation that medications may have served to restrict the range of responsiveness in the current study is indirectly

supported by the findings of a significant drug effect and Type x Drug interaction for ACTH. Consistent with previous literature (Durel et al., 1985), beta-blockers, calcium-channel blockers, and both, were associated with reduced responsivity to the stressor in all subjects (drug main effect). Moreover, the significant Type x Drug interaction for ACTH indicated that the target drugs differentially affected Type A and Type B subjects as was predicted in the hypotheses. Type As in the no drug condition of the independent variable of drug group were more reactive (i.e., greater concentrations of ACTH) to the stressor than were Type As in the drug condition. The use of the target medications appeared to have less of an impact on Type Bs subjects because ACTH concentrations were similar regardless of the drug condition (i.e., drug or no drug). These data suggest, then, that the target drugs were more effective for Type A subjects. It should be noted that two important points make specific comparisons of the current data to past research findings difficult. First, previous research on the effects of pharmacologic interventions on stress have typically studied one drug at the time. The current study compared the effects of beta-blockers, calcium-channel blockers, and both of these combined to a group of patients taking neither of these drugs. Second, previous Type A reactivity research has, without question, neglected the investigation of ACTH. Given these considerations, it is difficult to compare the current findings with those of previous research. Nonetheless, these results, suggesting that ACTH reactivity to stress can be

reduced pharmacologically, can be considered as breaking new ground for future research. Some degree of caution is necessary in interpreting these results until future replications are conducted. However, the nature of the sample size ($N = 48$) and the degree of significance ($p < .003$) for the Type x Drug interaction would suggest that these results do not reflect spurious findings.

Several important research and clinical implications can be drawn from the drug main effect and the Type x Drug interaction obtained for ACTH. First from the standpoint of research, the current data suggest that the control and evaluation of the effects of various medications upon reactivity is necessary in future research. It is possible, based on the current data, that recent studies failing to find A-B differences (e.g., Kornfeld et al., 1985) may have resulted from an inadequate investigation of drug effects. From a clinical perspective, several implications can be drawn from the current study. One of the more exciting areas of recent Type A research involves the hypothesis that beta-blockers effectively modify Type A behavior to that more consistent with Type B behavior patterns (Durel et al., 1985). The current study provides indirect support for this hypothesis in that drugs had the effect of reducing the responsivity of the Type A subjects to that exhibited by Type B subjects. Further, if one accepts the evidence that Type A behavior is associated with an increased risk of coronary heart disease, then the use of drugs that could modify Type A behavior may decrease Type A related CHD

risk. There is evidence from the research of Brand et al. (1976) that a 31 per cent reduction of CHD risk would occur if the direct risk associated with Type A were eliminated. Therefore, the use of beta-blockers, given continued supportive research, may make a valuable contribution to reducing CHD. Knowledge that certain medications can substantially reduce the Type A individuals' response to a stressful situation (e.g., surgery) would also be useful information to the physician. This knowledge would be particularly beneficial if the stressor carried with it dangerous side effects (eg., coronary artery bypass grafting). In situations such a major surgery, the use of medications to reduce anxiety may lead to a lesser incidence of post-operative complications. Investigations of the combined efficacy of drugs and well-known psychological methods of anxiety management could be another avenue for research.

A significant Type x Day interaction was also found for SBP. Inconsistent with previous research (Krantz et al., 1982; Dembroski et al., 1977) and with the current hypotheses, SBP of Type A subjects did not differ from Type B subjects when exposed to the stressor (day 1). It is possible that the use of drugs in the greater majority of patients served to restrict the range of SBP responsiveness to the stressor. Inconsistencies with previous research (Jennings et al., 1984, Friedman et al., 1960) and the current hypotheses were also found for SBP on day 2. Research would suggest that Type As and Bs should be similar during periods of rest. This was not supported by the current data. It is

unclear why Type A subjects, as compared to Type B subjects, demonstrated lower levels of SBP on day 2.

Extreme Sample

A very different pattern of results was observed using the extreme sample. In fact, the only significant findings for this sample were day effects for state anxiety and HR. No main effects for behavior type were revealed. This is inconsistent with previous findings that extreme type As are more reactive to stress than their non-extreme Type A counterparts (Krantz et al., 1984). Two points should be noted regarding this analyses. First, it was not possible to investigate medications in this analysis. As has been suggested previously, the failure to investigate the effects of medications may obscure A-B differences. Second, extreme Type As and Bs in the current study were identified in a slightly different manner than is typically used in the current literature. Typically, extreme scorers are those falling one standard deviation above (extreme Type A) or below (extreme Type B) the mean on the Overall Type A score of the JAS. Upper and lower quartiles were used in the present study. Whether or not this modest difference in identifying extreme scorers accounted for the lack of significant A-B differences is unknown and remains a question for future research. It should be noted, however, that obtaining large sample sizes of extreme subjects is rather problematic for the researcher interested in this area.

Correlational Data

A review of the Pearson correlation matrix suggests several

points. First, the correlation of each dependent variable with itself (e.g., ACTH on day 1 correlated with ACTH on day 2) was highly significant suggesting stability of each dependent measure across both days of measurement. As has been suggested, the use of medications may have served to provide a ceiling effect on arousal. More importantly, perhaps, were the lack of significant correlations among the biochemical, cardiovascular, and self-report measures of arousal. The lack of correlation among different response channels of anxiety (i.e., physiological, self-report, and motoric responses) is a reliable finding in the anxiety literature (Neitzel & Bernstein, 1981). Termed "response desynchrony," the lack of correlation among response channels of anxiety is thought to occur because the display of anxiety is a result not only of the eliciting stimulus but of other factors as well. For example, in the present study, it is likely that, although a subject may have felt subjective distress as a result of the impending catheterization, the distress may not have been reflected in the self-report measures because the subject may not have wanted to appear distressed to the experimenter. There was also a lack of correlation among the physiological measures. Although SBP and DBP were significantly associated with one another, neither were found to be associated with HR. These findings are consistent with previous research as it is not uncommon for different physiological measures to correlate poorly with one another (Lacey, 1967). The occurrence of desynchrony among response channels as well as within channels has number of

implications. First, the investigation of anxiety or arousal requires multiple assessment methods. Second, it is probably best to avoid statistical methods of analysis that assume intercorelations among dependent variables (e.g., multivariate approaches).

Summary and Future Directions

The major contribution of the current study was the finding that medications (i.e., beta-blockers, calcium-channel blockers, and both combined) were associated with significantly reduced responsivity to stress in Type A cardiac patients. The implications of this finding were discussed and the continued control for and evaluation of the effects of medications upon reactivity was emphasized.

Based on the current results, a number of future research directions are implicated. First, systematic investigation regarding the influence of age upon reactivity would be useful. Second, a more thorough analysis of the effects of various drug groups (e.g., beta-blockers, calcium channel blockers, nitrates) upon reactivity is necessary before definitive statements can be made. Finally, future reactivity research is likely to benefit from more frequent sampling of dependent measures, particularly when endocrinological responses are assessed.

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Appendix A

INFORMED CONSENT AGREEMENT

I, _____, do hereby consent to participate in the study entitled "The Type A Behavior Pattern and ACTH Response to the Stress of Cardiac Catheterization", the purpose of which is to investigate how different individuals respond to cardiac catheterization. I understand that I have been scheduled for cardiac catheterization on the advice of my physician and that I am not being catheterized because of my participation in the study. As such I will be signing a separate consent agreement to undergo cardiac catheterization. Other than procedures to be described below, my treatment while hospitalized will be identical to that of any patient hospitalized for cardiac catheterization.

Ms. Marie Veitia, M.S., psychology intern under the supervision of Dr. John Roitzsch, has explained to me verbally the procedures, as described below, and I fully understand the following:

A. Procedure

1. Upon my consent to participate in the study, I will be asked to complete a Personal Information Form which will ask for demographic information (age, education, occupation) and for information about the presence of traditional risk factors (smoking history, family history of cardiovascular disease, etc.).

2. I agree to complete the following questionnaires at the times designated below. I understand that Ms. Veitia will show me these forms as they are described.

a) Activity Survey (to be completed the evening before the catheterization)

b) Anxiety Inventory (to be completed the morning prior to catheterization and the morning after)

c) Mood Inventory (to be completed the morning prior to catheterization and the morning after)

All of these questionnaires are brief, each requiring 10-15 minutes or less to complete.

3) I agree to allow trained nursing personnel to draw blood with a needle from a vein in my arm on two occasions- the morning prior to catheterization and the morning after. Approximately 10 cc (about two teaspoonfuls) of blood will be drawn during each of the two blood drawing times. This blood will be analyzed for chemicals (hormones) that occur during stress.

4) I agree to allow Ms. Veitia to examine my medical record to obtain other pertinent information (for example, vital signs such as blood pressure and heart rate).

B. Duration : Upon my discharge from MUH, my participation in the study will be completed.

C. Possible Discomforts and/or Risks : The procedure to be followed in the study are standard and non-experimental. When blood is drawn from my arm, I may feel a slight pain and the spot from which the blood is taken may be temporarily bruised. There is a slight chance of inflammation of the vein and/or a blood clot formation, but this is extremely rare. There are no risks associated with taking the psychological questionnaires included in the study. I understand that there are risks associated with the cardiac catheterization and these will be explained to me on a separate consent agreement to undergo this procedure, since undergoing the catheterization itself has been scheduled for medical reasons and not for purposes of this study.

D. Possible Benefits : I understand that the study may not benefit me directly, but will help scientists obtain new knowledge that may benefit patients in the future. I understand that, if desired, an interpretation of my psychological testing will be made available to me upon completion of the study. If I would like appropriate referral, this will be made available to me.

Ms. Marie Veitia has agreed to answer any inquiries that I may have concerning the procedures and has informed me that I may also contact the Medical University of South Carolina Institutional Review Board for Human Research (803/792-4148) directly concerning patient rights. This board administers the agreement with the United States Department of Health and Human Services covering the protection of human subjects. I understand that in the event of any injury resulting from the research procedures to the participant, reasonable medical treatment not otherwise covered by third party payments will be available free through the Medical University; financial compensation is not available for medical treatment elsewhere, loss of work, or other expenses. I may contact the Medical University of S.C. Hospital Medical Director(803/792-3932) concerning medical treatment.

I understand that the participant's records of participation in this study are not accessible to the general public and confidentiality will be maintained. Information that may be gained from this study will be used only for research and educational purposes. Information may be published with the permission of the principal investigator in medical journals, but the participant's identity will not be revealed. However, identifying information will be available to monitors from the MUSC I.R.B. for Human Research and the U.S. Food and Drug Administration.

It is understood that participation is totally voluntary, and I may choose not to participate. I also understand that I am free to withdraw my consent and discontinue participation at any time. Discontinuation will in no way jeopardize the participant's ability to receive treatment now or in the future at this institution.

I will receive a copy of the informed consent after it has been read, understood, and signed.

DOCTOR OBTAINING CONSENT

SIGNATURE OF
PARTICIPANT

WITNESS

WITNESS

DATE OF CONSENT

SIGNATURE OF LEGAL
GAURDIAN
(if applicable)

Appendix B

PERSONAL INFORMATION FORM

NAME _____ DATE _____
 DATE OF BIRTH _____ HEIGHT _____ WEIGHT _____

EDUCATION: _____ DID NOT GRADUATE HIGH SCHOOL
 _____ GRADUATED HIGH SCHOOL
 _____ COMPLETED TRADE SCHOOL
 _____ SOME COLLEGE _____ NO. OF YEARS
 _____ GRADUATED FROM FOUR YEAR COLLEGE
 _____ POSTGRADUATE WORK _____ HIGHEST DEGREE

CURRENT EMPLOYMENT STATUS:

_____ FULL TIME
 _____ PART TIME _____ HOURS PER WEEK
 _____ UNEMPLOYED
 _____ RETIRED _____ YEAR
 _____ DISABLED _____ YEAR

DESCRIPTION OF PRESENT JOB:

Job Title _____ No. of years _____ Average
 hours per week _____

ACTIVITY LEVEL: Which category listed below best describes
 your current activity? _____ Which best describes your
 activity in the past year? _____

1. Active: An athlete in training or a person who exercises at a level comparable to running at least 10 miles per week or a job involving heavy manual labor.
2. Moderately Active: Planned recreation, such as running, swimming, bicycling, at least three days a week or a job involving moderate activity, such as construction work or farming.
3. Light Activity: Gardening, fishing, walking a mile most days, or a job involving walking or frequent step climbing.
4. Sedentary: Only normal daily activities such as eating, sleeping, sitting, talking, attending school, or a sedentary job.

SMOKING HISTORY: Do you smoke now (cigarettes, pipe, cigars)?
 Yes, No

If cigarettes, how many packs per day? _____
 Total number of years smoked? _____

FAMILY HISTORY: Does anyone in your family have (or had)
 heart disease? _____ Whom? _____ Has anyone
 in your family ever died of heart disease? _____ At what age or
 ages? _____

ADDITIONAL RISK FACTORS:

☐ High blood pressure
☐ Diabetes
☐ High dietary intake of fat
☐ High cholesterol or triglycerides
☐ previous heart attack

WHAT ARE YOUR CURRENT MEDICATIONS? DOSAGE FOR HOW LONG?

(circle as many as apply)

NITRATES AND NITROGLYCERINS

Cardilate (oral)
 Diltrate (oral)
 Nitrong (oral)
 NitroBid (oral)
 Nitroglyn (oral)
 Isordil (oral)
 Sorbide (oral)
 IsoBid (oral)
 Duotrate (oral)
 Pentritol (oral)
 Peritrate (oral)
 Nitrospan (oral)
 Tridil (oral)
 Cardabid (oral)
 Nitrol Ointment
 NitroBid Ointment
 Nitrostat Ointment
 Nitrong Ointment
 Nitrodisc (transdermal)
 Nitro-Dur (transdermal)
 Transderm NTG (transderm)
 Susadrin Transmucosal tabs

BETA-BLOCKERS

Inderal (Propanolol HCL)
 Viskin (Pindolol)
 Blockadren (Timolol maleate)
 Corgard (Nadolol)
 Lopressor (Metropolol)
 Tenormin (Atenolol)

CALCIUM ENTRY BLOCKERS

Procardia (Nifedipine)
 Isoptin (Verapamil)
 Calan (Verapamil)
 Cardizem (Diltiazem HCL)

OTHERS:(Including psychiatric meds):

REASON FOR HEART CATH?

HAVE YOU EVER HAD A HEART CATH BEFORE? Y OR N If so, when?

HAVE YOU EVER HAD A CORONARY ARTERY BYPASS? Y OR N If so,
when?

Appendix C

Appendix C1: ACTH Statistical Summary Table for 2 x 2 x 2 ANOVA
with Repeated Measures on Day (N = 48)

Appendix C2: SBP Statistical Summary Table for 2 x 2 x 2 ANOVA
with Repeated Measures on Day (N = 60)

Appendix C3: DBP Statistical Summary Table for 2 x 2 x 2 ANOVA
with Repeated Measures on Day (N = 60)

Appendix C4: HR Statistical Summary Table for 2 x 2 x 2 ANOVA
with Repeated Measures on Day (N = 60)

Appendix C5: State Anxiety Statistical Summary Table for 2 x 2 x
2 ANOVA with Repeated Measures on Day (N = 60)

Appendix C6: TMD Statistical Summary Table for 2 x 2 x 2 ANOVA
with Repeated Measures on Day (N = 60)

Appendix C1

ACTH Statistical Summary Table for 2 x 2 x 2 (Behavior Type x Drug
Group x Day) ANOVA with Repeated Measures on Day (N = 48)

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
Type	1	2059.21	2.20	.14
Drug	1	4136.15	4.42	.04
Type x Drug	1	9003.73	9.63	.003
Day	1	2005.15	6.38	.01
Type x Day	1	747.91	2.38	.13
Drug x Day	1	117.39	0.37	.54
Type x Drug x Day	1	447.53	1.42	.24
<u>S</u> s (Type x Drug)	44	935.99		
Day x <u>S</u> s (Type x Drug)	44	314.16		

Appendix C2

SBP Statistical Summary Table for 2 x 2 x 2 (Behavior Type x Drug
Group x Day) ANOVA with Repeated Measures on Day (N = 60)

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
Type	1	234.57	0.56	.46
Drug	1	60.09	0.14	.71
Type x Drug	1	6.45	0.01	.91
Day	1	628.48	4.51	.04
Type x Day	1	890.77	6.39	.01
Drug x Day	1	43.55	0.31	.58
Type x Drug x Day	1	392.54	2.82	.10
<u>S</u> s (Type x Drug)	56	436.65		
Day x <u>S</u> s (Type x Drug)	56	139.42		

Appendix C3

DBP Statistical Summary Table for 2 x 2 x 2 (Behavior Type x Drug
Group x Day) ANOVA with Repeated Measures on Day (N = 60)

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
Type	1	159.26	1.09	.30
Drug	1	1.34	0.01	.92
Type x Drug	1	58.66	0.40	.53
Day	1	5.72	0.12	.73
Type x Day	1	92.36	1.94	.17
Drug x Day	1	16.97	0.36	.55
Type x Drug x Day	1	24.68	0.52	.47
<u>S</u> s (Type x Drug)	56	145.79		
Day x <u>S</u> s (Type x Drug)	56	47.70		

Appendix C4

HR Statistical Summary Table for 2 x 2 x 2 (Behavior Type x Drug
Group x Day) ANOVA with Repeated Measures on Day (N =60)

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
Type	1	163.41	1.06	.31
Drug	1	60.77	0.39	.53
Type x Drug	1	587.50	3.80	.06
Day	1	89.60	2.39	.13
Type x Day	1	34.96	0.93	.34
Drug x Day	1	34.23	0.91	.34
Type x Drug x Day	1	119.71	3.20	.08
<u>S</u> s (Type x Drug)	56	154.08		
Day x <u>S</u> s (Type x Drug)	56	37.43		

Appendix C5

State Anxiety Statistical Summary Table for 2 x 2 x 2 (Behavior
Type x Drug Group x Day) ANOVA with Repeated Measures on Day (N
 = 60)

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
Type	1	3.17	0.02	.88
Drug	1	0.83	0.01	.94
Type x Drug	1	56.68	0.39	.53
Day	1	148.23	3.97	.05
Type x Day	1	4.16	0.11	.74
Drug x Day	1	16.45	0.44	.51
Type x Drug x Day	1	2.82	0.08	.78
<u>S</u> s (Type x Drug)	56	146.10		
Day x <u>S</u> s (Type x Drug)	56	37.38		

Appendix C6

TMD Statistical Summary Table for 2 x 2 x 2 (Behavior Type x DrugGroup x Day) ANOVA with Repeated Measures on Day (N = 60)

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
Type	1	32.90	0.02	.88
Drug	1	233.92	0.15	.70
Type x Drug	1	89.21	0.06	.81
Day	1	556.90	2.03	.16
Type x Day	1	18.35	0.07	.80
Drug x Day	1	165.92	0.61	.44
Type x Drug x Day	1	26.43	0.10	.76
<u>S</u> s (Type x Drug)	56	1575.74		
Day x <u>S</u> s (Type x Drug)	56	274.25		

Appendix D

Appendix D1: ACTH Statistical Summary Table for 2 x 2 ANOVA with Repeated Measures on Day (N = 22)

Appendix D2: SBP Statistical Summary Table for 2 x 2 ANOVA with Repeated Measures on Day (N = 30)

Appendix D3: DBP Statistical Summary Table for 2 x 2 ANOVA with Repeated Measures on Day (N = 30)

Appendix D4: HR Statistical Summary Table for 2 x 2 ANOVA with Repeated Measures on Day (N = 30)

Appendix D5: State Anxiety Statistical Summary Table for 2 x 2 ANOVA with Repeated Measures on Day (N = 30)

Appendix D6: TMD Statistical Summary Table for 2 x 2 ANOVA with Repeated Measures on Day (N = 30)

Appendix D1

ACTH Statistical Summary Table for 2 x 2 (Behavior Type x Day)ANOVA with Repeated Measures on Day (N = 22)

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
Type	1	5.24	0.01	.94
Day	1	280.18	0.75	.40
Type x Day	1	278.20	0.74	.40
<u>S</u> s x Day (Type) 20		374.99		

Appendix D2

SBP Statistical Summary Table for 2 x 2 (Behavior Type x Day)ANOVA with Repeated Measures on Day (N = 30)

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
Type	1	928.27	2.66	.11
Day	1	224.27	1.77	.19
Type x Day	1	317.40	2.51	
Day x <u>S</u> s (Type) 28		126.37		

Appendix D3

DBP Statistical Summary Table for 2 x 2 (Behavior Type x Day)ANOVA with Repeated Measures on Day (N = 30)

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
Type	1	147.27	1.26	.27
Day	1	24.07	0.55	.47
Type x Day	1	19.27	0.44	.51
Day x <u>S</u> s (Type)	28	43.67		

Appendix D4

HR Statistical Summary Table for 2 x 2 (Behavior Type x Day) ANOVA
with Repeated Measures on Day (N = 30)

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
Type	1	60.00	0.39	.54
Day	1	166.67	4.30	.05
Type x Day	1	60.00	1.55	.22
Day x <u>S</u> s (Type)	28	38.76		

Appendix D5

State Anxiety Statistical Summary Table for 2 x 2 (Behavior Type x
Day) ANOVA with Repeated Measures on Day (N = 30)

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
Type	1	96.27	0.76	.39
Day	1	153.60	4.07	.05
Type x Day	1	0.60	0.02	.90
Day x <u>S</u> s (Type)	28	37.74		

Appendix D6

TMD Statistical Summary Table for 2 x 2 (Behavior Type x Day)ANOVA with Repeated Measures on Day (N = 30)

<u>Source</u>	<u>df</u>	<u>MS</u>	<u>F</u>	<u>p</u>
Type	1	64.07	0.04	.84
Day	1	405.60	2.86	.10
Type x Day	1	395.27	2.79	.11
Day x <u>S</u> s (Type)	28	141.90		

CURRICULUM VITAE
(Revised September 1986)

Marie C. Veitia, M.S. **BIRTHDATE:** January 18, 1957

PRESENT HOME ADDRESS:

6145 Country Club Drive
Huntington, WV 25705

TELEPHONE NUMBER:

304/736-0566

SOCIAL SECURITY NUMBER:

260-96-3567

LANGUAGES SPOKEN:

English, Spanish

CURRENT POSITION:

Assistant Professor of Medical Psychology
Department of Psychiatry
Marshall University School of Medicine
Huntington, WV 25701
September 1986 - present

EDUCATION:

1974-1976	Anderson College Anderson, SC Received A.A., May 1976
1976-1977	Furman University Greenville, SC
1977-1981	University of New Orleans New Orleans, LA Major (Undergraduate): Psychology Major (Graduate): Physiological Psychology Received B.A., May 1979 Received M.S., December 1981

1981-1986 Louisiana State University
Baton Rouge, LA
Major: Clinical Psychology
Received Ph.D., September 1986

CLINICAL EXPERIENCE:

9/85 - 8/86 Veterans Administration Medical Center
Alcohol Dependence Treatment Program
Position: Psychology Technician
Supervisor: John C. Roitzsch, Ph.D.
Description: Employed on a part-time
basis while completing my dissertation.
Chief responsibilities were outpatient
treatment with chronic alcoholics,
emergency consultations, and outpatient
treatment groups.

PRE-DOCTORAL INTERNSHIP:

9/84 - 8/85 Medical University of South Carolina/
Veterans Administration Medical Center,
Charleston, SC (APA approved)
Position: Psychology Intern
Preceptor: John C. Roitzsch, Ph.D.

Description of Training:

Alcohol Dependence Treatment Program
(VAMC): Individual assessment and
treatment with inpatient and outpatient
alcoholics; administration and
interpretation of testing; conducted
problem-solving and assertiveness
groups; emergency on call psychiatric
services.

Supervisor: John C. Roitzsch, Ph.D.

Mental Hygiene Clinic (VAMC):
Outpatient therapy with individuals
and families; behavioral,
psychological, and neuropsychological
assessments; supervision of medical

students learning interviewing and diagnostic skills; emergency, on call psychiatric services.

Supervisor: Julian Libet, Ph.D.

Adult Inpatient Unit (MUSC):

Psychological assessments including personality, intellectual, and neuropsychological; individual assessment and treatment of inpatient adults; co-leader of group therapy sessions; conducted training seminar for medical students; co-leader of stress management group.

Supervisor: Sid Jordan, Ph.D.

Consultation-Liaison Service (MUSC):

Responded to consults and provided psychological follow-up care to patient at MUSC and Charleston Memorial Hospital; established liaison with Physical Medicine and Rehabilitation Unit (MUSC); conducted support group for patients with spinal cord injuries.

Supervisor: Connie L. Best, Ph.D.

CLINICAL PRACTICA:

8/83 - 8/84

Earl K. Long Memorial Hospital
Medical Consultation/Liaison Service
Baton Rouge, LA
Position: Medical Psychology Trainee
Supervisor: Phillip J. Brantley, Ph.D.
Description of Training: Behavioral medicine techniques, traditional and behavioral assessment, and brief outpatient psychotherapy.

- 8/82 - 9/83 Psychological Services Center
Louisiana State University
Baton Rouge, LA
Position: Adult Psychology Trainee
Supervisor: William F. Waters, Ph.D.
Description of Training: Traditional
and behavioral assessment techniques
and adult psychotherapy involving
marital and sexual dysfunction,
depression, anxiety, etc.
- 9/81 - 8/82 Earl K. Long Memorial Hospital
Family Practice Unit
Baton Rouge, LA
Position: Medical Psychology Trainee
Supervisor: Phillip J. Brantley, Ph.D.
Description of Training: Child and
adult behavioral assessment and
psychotherapy.
- 1/81 - 5/81 Southeast Louisiana Hospital
Mandeville, LA
Position: Practicum Student
Supervisor: Suzanne Hill, Ph.D.
Description of Training: Group
psychotherapy with adolescent
females, individual therapy with
autistic male adolescents.

CONSULTATION EXPERIENCE:

- 9/85 -11/85 Stress Management Workshop for City
Employees of
Charleston, South Carolina. Co-
leader of 8-week program (one
session/week) sponsored by the
MUSC/The Citadel Cardiac
Rehabilitation and Adult Fitness
Program.
- Supervisor: John C. Roitzsch, Ph.D.

9/84 - 8/85

Cardiac Rehabilitation Program
Medical University of South
Carolina/The Citadel
Description: Assisted Dr. John
Roitzsch with his consultation
responsibilities for the program,
including assessment of psychosocial
risk for heart disease and
presentation of topics related to
stress.

9/83 - 8/84

Department of Rehabilitation
U.S. Public Health Service Hospital
Carville, LA
Supervisor: Phillip J. Brantley, Ph.D.
Description of Training: Traditional
and behavioral assessment and brief
psychotherapy with Spanish-speaking
Hansen's Disease patients; Conducted
depression management group with
Spanish-speaking female patients with
Hansen's Disease.

RESEARCH EXPERIENCE:

1/85 - 8/86

Charleston Veterans Administration Medical Center
Supervisors: Phillip J. Brantley, Ph.D. & John C.
Roitzsch, Ph.D.
Dissertation: The Type A Behavior
Pattern and the ACTH Response to
Cardiac Catheterization.

9/84 - 8/86

Charleston Veterans Administration
Medical Center
Supervisor: John C. Roitzsch, Ph.D.
Member of research team investigating
coronary illness and chronic
alcoholism.

8/83 - 8/84

Earl K. Long Memorial Hospital
Supervisors: Phillip J. Brantley,
Ph.D. & J. D. Martin, M.D.
Member of interdisciplinary research
team investigating the management of
severe bronchial asthmatics.

- 1/83 - 8/84 Louisiana State University
 Supervisor: Frank M. Gresham, Ph.D.
 Member of research team investigating
 social skills in normal adolescents.
- 2/82 - 2/84 Louisiana State University
 Supervisor: Donald A. Williamson, Ph.D.
 Member of a research team
 investigating the assessment and
 treatment of bulimia.
- 6/82 - 8/82 Employed as a research assistant for
 a grant: Use of social competence
 measures to facilitate parent and
 teacher involvement and nonbiased
 assessment. United States Department
 of Education (Daniel K. Reschly and
 Frank M. Gresham - co-investigators).
- 1/81 - 5/82 University of New Orleans
 Supervisor: Sarah Moody Thomas, Ph.D.
 Member of a research team
 investigating social skills in
 institutionalized adolescent males.
- 12/79-12/81 University of New Orleans
 Supervisor: Bruce M. King, Ph.D.
 Completed an animal study examining
 the hypothalamic control of hunger
 and thirst.

CONVENTION PRESENTATIONS:

Gresham, F.M., Bruce, B.K., & Veitia, M.C. (1983).
Convergent discriminant validity in the assessment of
 adolescents' social skills. Paper presented at the
 combined meeting of the Association for Advancement of
 Behavior Therapy and World Congress on Behavior Therapy,
 Los Angeles, CA.

Gresham, F.M., Bruce, B.K., Veitia, M.C., & Jones, G.R.
 (1985). Sex bias in peer assessment of adolescent social
 skills. Paper presented at the meeting of the
 Southeastern Psychological Association, Atlanta, GA.

Malcolm, R.J., Veitia, M.C., Roitzsch, J.D., Giacinto, J.P., & Scott, P. (1985). The effect of detoxification on blood pressure. Paper presented at the meeting of Research Society on Alcoholism, Isle of Palms, SC, (abstract published).

Roitzsch, J.C., Giacinto, J.P., Malcolm, R.J., Veitia, M.C., & AuBuchon, P.G. (1985). Alcoholism and achievement anxiety. Paper presented at the meeting of the Research Society on Alcoholism, Isle of Palms, SC, (abstract published).

Roitzsch, J.C., Giacinto, J.P., Veitia, M.C., & Christopher, S. (1985). The relationship between Type A behavior and hostility in cardiac patients: Does "A" stand for anger? Paper presented at the meeting of the Southeastern Psychological Association, Atlanta, GA.

Veitia, M.C., Brantley, P.J., Callon, E.B., Buss, R.R., & Sias, C.R. (1983) Assessing the impact of psychological intervention on medical utilization in a subsidized health care setting. Paper presented at the combined meeting of the Association for Advancement of Behavior Therapy and World Congress on Behavior Therapy, Los Angeles, CA.

Veitia, M.C., Bruce, B.M., & Levine, S. (1981) Drinking and stress responses to an osmotic challenge in rats with lateral hypothalamic lesions. Paper presented at the meeting of the Southwestern Psychological Association, Houston, TX.

Veitia, M.C., Giacinto, J.P., & Roitzsch, J.C. (1985) The display of anger in alcoholics: Active aggressive or passive aggressive? Paper presented at the meeting of the Southeastern Psychological Association, Atlanta, GA.

Williamson, D.A., Kelley, M.L., Davis, C.J., Ruggiero, L., & Veitia, M.C. (1983) Psychophysiology of bulimia. Paper presented at the combined meeting of the Association for Advancement of Behavior Therapy and World Congress on Behavior Therapy, Los Angeles, CA.

PUBLICATIONS:

Bahlinger, V., Brantley, P.J., Heroman, M.W., Mandrigal, D., & Veitia, M.C. (1985). Psychosocial stress in Hansen's Disease: A comparison with other chronic diseases. International Journal of Leprosy and Other Biobacterial Diseases, 53, 251-254.

Brantley, P.J., Veitia, M.C., Callon, E.B., Buss, R.R., & Sias, C.R. (in press) Effects of brief psychological intervention on medical overutilization in family practice patients. Journal of Family Practice.

Bruce, B.K., King, B.M., Phelps, G.R., & Veitia, M.C. (1981). Effects of adrenalectomy and corticosterone administration on hypothalamic obesity in rats. American Journal of Physiology, 243, 152-157.

Faulstich, M.E., Gresham, F.M., McAnulty, D.P., Veitia, M.C., Moore, J.R., Bernard, B., Waggoner, C.D., & Howell, R.I. (in press). Factor structure of the WAIS-R for an incarcerated population. Law and Psychology Review.

Gresham, F.M., Bruce, B.K., Veitia, M.C., & Jones, G. (in press). Same-sex, cross-sex, and age differences in peer assessment of adolescent social skills. Journal of Early Adolescence.

Gresham, F.M., Lemanek, K., & Veitia, M.C. (1982). Social Skills: Principles, procedures, and practices: A videotape. Des Moines, Iowa: Iowa Department of Public Instruction.

McAnulty, D.P., Gresham, F.M., Faulstich, M.E., Veitia, M.C., Howell, R.I., Waggoner, C.D., Moore, J.R., & Bernard, B. (in press) Two- and four-subtest WAIS-R short forms in a prison population. Journal of Clinical Psychology.

Veitia, M.C., King, B.M., & Levine, S. (1982). Adrenocortical response to hypertonic saline in rats with lateral hypothalamic lesions. Journal of Comparative and Physiological Psychology, 96, 846-849.

Williamson, D.S., Kelley, M.L., Davis, C.J., Ruggiero, L., & Veitia, M.C. (1985). Psychophysiology of Bulimia. In T. Wilson & S. Rachman (Eds.), Advances in Behavior Research and Therapy, pp. 163-172. Oxford, England: Pergamon Press.

GRANTS AWARDED:

Awarded \$3,200.00 grant from Stuart Pharmaceuticals to conduct dissertation.

RESEARCH IN PROGRESS:

Assessment of reactivity to stress in Type A cardiac patients

Preventive cardiology in medical education.

REFERENCES:

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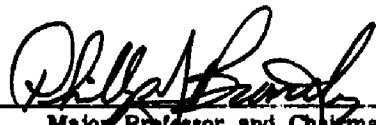
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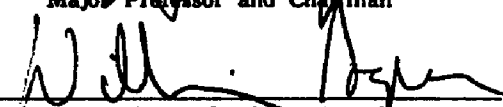
Candidate: Marie C. Veitia

Major Field: Psychology


Title of Dissertation: The Type A Behavior Pattern and ACTH Response to the Stress of Cardiac Catheterization


Approved:

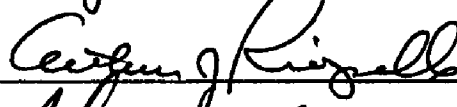

Major Professor and Chairman



Dean of the Graduate School

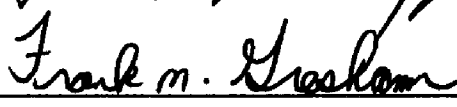
EXAMINING COMMITTEE:











Date of Examination:

September 26, 1986